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PREVALENCE OF COMMUNICABLE DISEASES IN THE UNITED STATES

April 21–May 18, 1940

The accompanying table summarizes the prevalence of eight important communicable diseases, based on weekly telegraphic reports from State health departments. The reports from each State are published in the Public Health Reports under the section "Prevalence of disease." The table gives the number of cases of these diseases for the 4-week period ended May 18, 1940, the number reported for the corresponding period in 1939, and the median number for the years 1935–39.

With the exception of influenza, the incidence during the 4 weeks ended May 18 of all of the eight communicable diseases under consideration was again below the median expectancy for the period.

Influenza.—While the number of cases of influenza (5,650) was only about 50 percent of the number reported for this period in 1939, the incidence was about 15 percent in excess of the 1935–39 median figure for this period. The disease was most prevalent in the South Atlantic and West South Central regions. In the South Atlantic region the number of cases (2,012) was more than twice the average incidence for preceding years, but in the West South Central region the number (1,792) was slightly below the seasonal expectancy. While the number of cases in the Mountain region was not large, it was almost twice the median figure for the period; in all other regions the situation was favorable.

DISEASES BELOW MEDIAN PREVALENCE

Diphtheria.—The incidence of diphtheria was the lowest on record for this period. For the 4 weeks ended May 18 there were 927 cases reported as compared with 1,221, 1,486, and 1,544 cases for the corresponding period in 1939, 1938, and 1937, respectively. The current incidence is less than 60 percent of the 1935–39 median figure for this period.

Measles.—The number of cases (44,682) of measles reported during the current period was about 70 percent of the number reported for the corresponding period in 1939 (approximately 62,000 cases), which

figure also represents the 1935-39 median figure for this period. The West South Central and Mountain regions reported rather significant increases over the normal seasonal expectancy, but in all other regions the incidence was comparatively low.

Number of reported cases of eight communicable diseases in the United States during the 4-week period Apr. 21-May 18, 1940, the number for the corresponding period in 1939, and the median number of cases reported for the corresponding period 1935-39¹

Division	Current period	1939	5-year median	Current period	1939	5-year median	Current period	1939	5-year median	Current period	1939	5-year median
	Diphtheria			Influenza ²			Measles ³			Meningococcus meningitis		
United States ¹	927	1, 221	1, 544	5, 650	10, 725	4, 939	44, 682	61, 913	61, 913	189	154	504
New England.....	26	31	47	12	322	30	5, 193	9, 550	9, 550	9	11	15
Middle Atlantic.....	187	242	314	88	82	69	8, 123	9, 231	19, 646	48	44	82
East North Central.....	144	273	289	444	617	617	6, 619	5, 701	9, 189	11	20	63
West North Central.....	84	75	118	71	271	271	4, 077	5, 269	5, 269	14	5	35
South Atlantic.....	152	177	244	2, 012	3, 796	926	2, 817	9, 119	6, 126	28	25	116
East South Central.....	58	81	124	517	1, 753	664	1, 559	1, 209	2, 110	32	14	52
West South Central.....	159	159	237	1, 792	3, 016	1, 842	5, 873	3, 656	2, 719	19	23	23
Mountain.....	50	72	72	453	492	229	4, 054	3, 956	3, 412	23	7	11
Pacific.....	67	111	119	261	376	376	6, 367	14, 222	9, 618	5	5	20
	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
United States ¹	66	149	78	19, 830	15, 980	24, 641	280	1, 229	1, 142	415	521	532
New England.....	1	1	3	1, 195	1, 094	1, 417	0	0	0	29	20	20
Middle Atlantic.....	7	6	6	7, 653	4, 606	6, 574	0	1	0	80	59	64
East North Central.....	7	8	9	7, 004	6, 236	8, 241	35	354	226	75	71	71
West North Central.....	5	3	3	1, 106	1, 445	3, 064	107	440	463	19	20	26
South Atlantic.....	8	95	15	819	573	767	7	5	5	58	89	106
East South Central.....	5	9	7	638	411	265	28	29	7	48	65	54
West South Central.....	6	11	11	207	299	426	51	243	61	57	119	124
Mountain.....	5	2	2	437	454	525	32	59	128	21	30	30
Pacific.....	22	14	14	771	862	1, 034	20	98	172	28	42	42

¹ 48 States. Nevada is excluded and the District of Columbia is counted as a State in these reports.

² 44 States and New York City.

³ 47 States. Mississippi is not included.

Meningococcus meningitis.—The incidence of meningococcus meningitis was slightly above that reported for the corresponding period in 1939, but in relation to the preceding 5-year average it was relatively low, the number of cases (189) for the current period being less than 40 percent of the average figure (504 cases) for this period. Of the 23 cases reported from the Mountain region, 19 occurred in New Mexico; the 1935-39 median figure for New Mexico for this period is only 5 cases.

Poliomyelitis.—For the 4 weeks ended May 18 there were 66 cases of poliomyelitis reported, as compared with 149, 64, and 78 cases for the corresponding period in 1939, 1938, and 1937, respectively. The Pacific region reported a few more cases than might normally be expected at this time, but in all other regions the situation was favorable.

Scarlet fever.—The number of cases (19,830) of scarlet fever reported for the current period represented an increase of approximately 20 percent over the number reported for the corresponding period in 1939, but the incidence remained well below the 1935–39 median incidence for this period (approximately 25,000 cases). Increases over the normal seasonal incidence were reported from the Middle Atlantic, South Atlantic, and East South Central regions, but in all other regions the incidence was below the seasonal expectancy.

Smallpox.—The incidence of smallpox was the lowest on record for this period. There were 280 cases reported, as compared with 1,229, 1,571, and 1,142 cases for the corresponding period in 1939, 1938, and 1937, respectively. Alabama reported 22 of the total of 28 cases reported from the East South Central region.

Typhoid fever.—The number of cases (415) of typhoid fever was the lowest reported for this period in the 12 years for which these data are available. The North Atlantic and East North Central regions reported some increase over the seasonal expectancy, but in other regions the incidence was relatively low. The South Atlantic and West South Central regions reported very significant declines from the 1935–39 median figures for this period.

MORTALITY, ALL CAUSES

The average mortality rate from all causes in large cities for the 4 weeks ended May 18, based on data received from the Bureau of the Census, was 11.4 per 1,000 inhabitants (annual basis). The rate was slightly higher than the rate for the corresponding period in each of the 2 preceding years. The 1935–39 average rate for this period was 11.8.

DISINSECTIZATION OF AIRCRAFT

By C. L. WILLIAMS, *Assistant Surgeon General, United States Public Health Service*

For some years, the Public Health Service has been actively engaged in the development of methods for destroying insects, particularly mosquitoes, on aircraft from foreign ports. Three specific objects have been aimed at: To prevent the introduction of mosquitoes infected with yellow fever from South American ports; to prevent the introduction of *Anopheles gambiae* from eastern South America into the southern part of the United States; and to prevent the introduction of any *Anopheles* from the west coast of the United States into the Hawaiian Islands.

In two papers published in 1935,¹ Williams and Dreessen describe the problem presented and recommend the use of a concentrated

¹ The destruction of mosquitoes in airplanes. By C. L. Williams and W. C. Dreessen. Pub. Health Rep., vol. 50, No. 20, May 17, 1935, pp. 663–671. A nonflammable pyrethrum spray for use in airplanes. By C. L. Williams and W. C. Dreessen. Pub. Health Rep., vol. 50, No. 41, Oct. 11, 1935, pp. 1401–1404.

pyrethrum spray which had been shown to be highly toxic to mosquitoes while practically innocuous to human beings. Based on this work, all planes coming from South America during the past few years have been sprayed throughout the fuselage while in flight en route from the last foreign stop to a United States port. The spraying has been done with a hand sprayer by the steward, the ventilating system of the airplane being cut off for a period of 10 minutes.

Careful inspections of airplanes on arrival at Miami, Fla., have shown that very few live mosquitoes have been brought into that port since the spraying in flight was instituted. Despite this, however, it is felt that the method is not entirely satisfactory for several reasons, the more important being that the spraying is not accomplished under the surveillance of a disinterested Government official, that the presence of passengers renders it difficult to direct the spray adequately into all of the remote recesses of the fuselage, and that certain compartments, such as the space under the limber boards, cannot be readily opened and sprayed in flight.

The airplanes traveling from northern South America, the Canal Zone, and Mexico City to Brownsville, Tex., were for some time sprayed while resting on the ground overnight at Mexico City and again while on the ground at Tampico. Careful inspection of these on arrival at Brownsville showed very few live mosquitoes, and apparently the method was a successful one.

Airplanes departing from San Francisco for Honolulu have been sprayed while resting on the water with the passengers aboard immediately before departure. This spraying has been done by a representative of the Public Health Service, and, it is believed, has been quite thorough in practically all instances. On arrival at Honolulu the discovery of live mosquitoes has been rare.

It has appeared that probably the best protection would be afforded by a very careful and thorough spraying of the interior of airplanes at a point removed both from infected territory and from the nearest United States port. It appeared that thereby a definite barrier could be set up against mosquitoes coming from infected areas, although it might not prevent the transportation of mosquitoes infesting the port at which the spraying itself was done, and which might come aboard following disinsectization. As long as such mosquitoes were either not infected or were not of a species that it was desired to keep out of the country, their transportation would be a matter of no quarantine significance.

With these considerations in mind, the Public Health Service has adopted, for the control of mosquitoes carried by aircraft from the eastern coast of South America, a method of spraying airplanes on the water at Port of Spain, Trinidad. This spraying is very carefully done with a power apparatus under the surveillance of a Public Health

Service inspector, who certifies that the spraying was properly performed. The procedure is as follows: A trained member of the Pan American Airways' ground personnel at Port of Spain carries aboard the plane an air-pressure sprayer which has been especially developed for the purpose, and after closing all openings to the outside and opening up all spaces inside the fuselage, including the bilges (the limber boards being raised), he proceeds to spray thoroughly the entire interior of the aircraft. The sprayer used permits accurate dosage, and an amount of insecticide is used double that which has been shown by experiment to be sufficient to kill 100 percent of exposed mosquitoes. As soon as all of the insecticide has been sprayed, the man operating the apparatus leaves the airplane, closing the hatch behind him, and the airplane is kept closed for 10 minutes. At the end of the 10 minutes, the airplane is opened and the crew goes aboard and begins preparations for departure. In a few minutes, the passengers come aboard, and within one-half hour from the time the spraying was begun, the airplane takes off.

By this method, it is felt that any mosquitoes that may have been brought by an airplane from South America are destroyed. It is true, of course, that mosquitoes infesting Trinidad may enter the plane after it is opened following disinsectization, even though the period before departure is only about 15 or 20 minutes. Mosquitoes entering at Trinidad, however, are not of present quarantine significance. Furthermore, tests of this method at Miami under laboratory conditions indicate that the residual insecticide in the aircraft to some extent repels mosquitoes that may attempt to enter and probably kills most of those that do enter.

At present, most of the airplanes leaving Trinidad for the United States proceed nonstop to San Juan, P. R. On arrival at that port, they are carefully inspected to determine whether any live mosquitoes are aboard. Between San Juan and Miami, stops are generally made at San Pedro de Macoris in the Dominican Republic, Port au Prince in Haiti, and Antilla in Cuba. None of these points is believed infested with mosquitoes that are of present quarantine significance. On arrival at Miami, the airplane is again carefully inspected to determine the presence of live mosquitoes. When sufficient data have been gathered, the results of these inspections will be published.

A NEW INSECTICIDE SPRAYER FOR AIRCRAFT

One of the difficulties in spraying aircraft has been to secure a sprayer with which relatively small amounts of insecticide could be accurately measured while spraying was in progress. This is necessary because most of the large aircraft are divided into compartments, and it is desired to introduce reasonably accurate doses into each

compartment. Another difficulty with air-pressure sprayers available has been that, upon closure of the air valve, some of the insecticide dripped from the nozzle. A third difficulty has been to secure a spray sufficiently fine that it might be regarded as dry, that is, that it would not deposit on walls and fabrics.

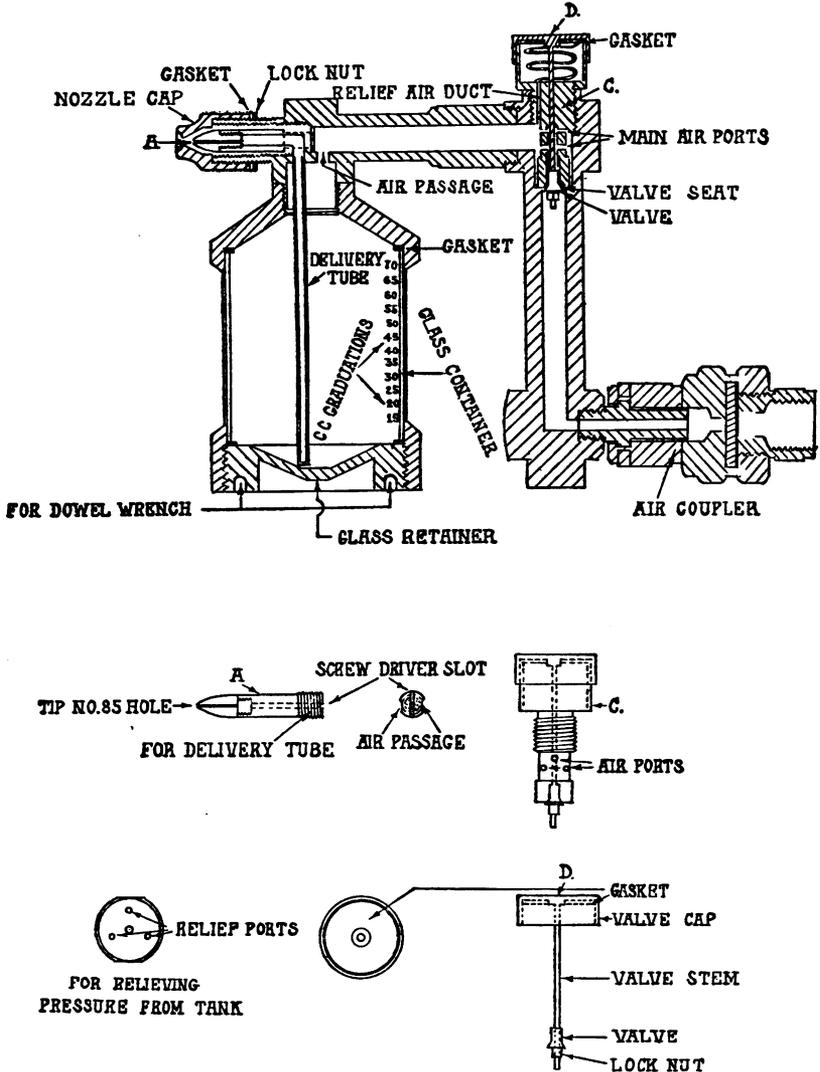


FIGURE 1.—Diagrammatic drawing showing construction of sprayer.

With these considerations in mind, Sanitary Engineer H. A. Johnson, about 3 years ago, designed a sprayer in which were incorporated a relatively small reservoir for the insecticide, an air valve that included a bleeder tube to relieve pressure on the insecticide when the air valve was closed, and a very fine spray orifice for the insecticide.

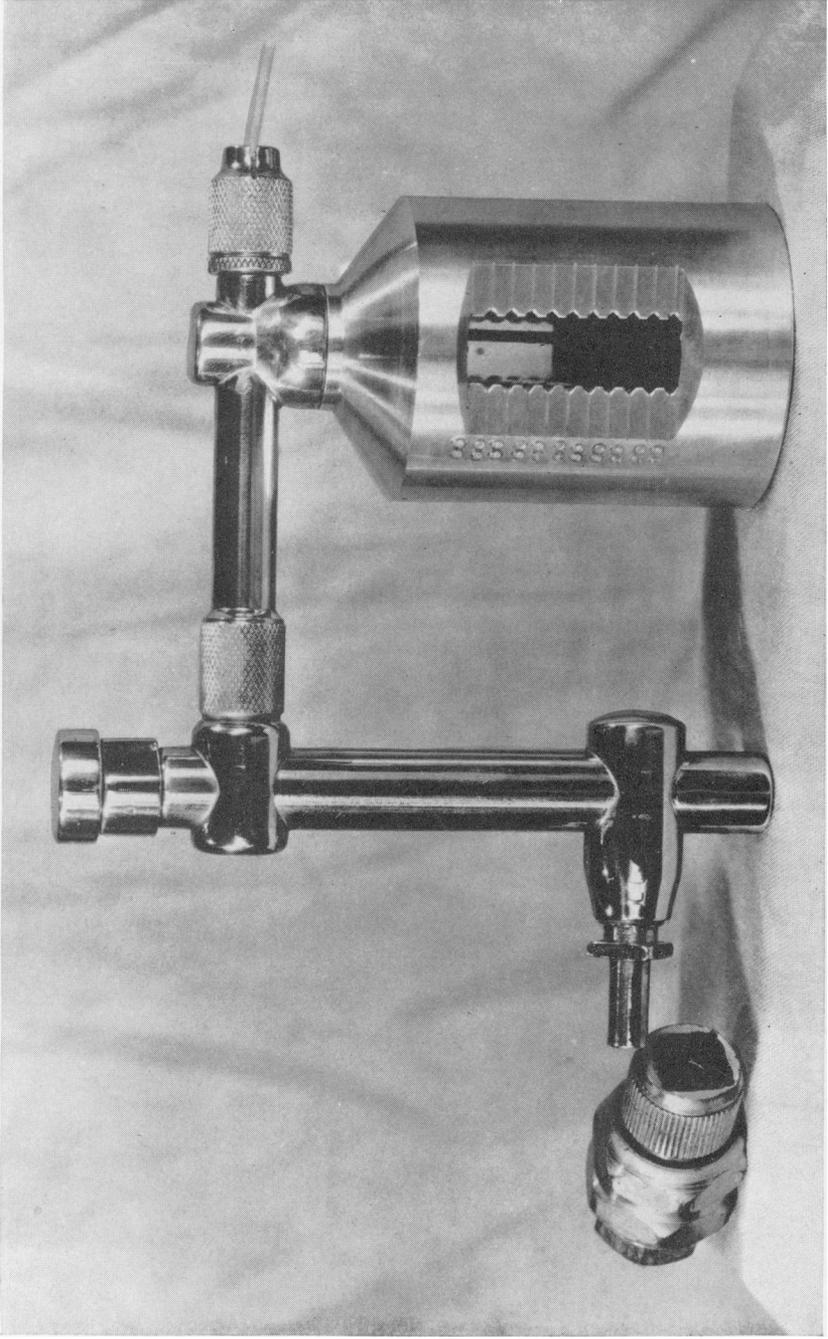


FIGURE 2.—The finished sprayer.



FIGURE 3.—The sprayer in use in an airplane.

Due to a change of duties, Mr. Johnson was not able to complete the development of this apparatus.

Utilizing Mr. Johnson's plans, Passed Assistant Surgeon G. L. Dunnahoo developed this sprayer for practical use and carried it with him to Trinidad when the spraying procedure described above was instituted. It is this sprayer that is now in use for spraying airplanes at Trinidad, and it is expected that it will be put into use for similar spraying of airplanes at other points in the Caribbean area, as well as in Mexico and on the west coast of the United States.

The first essential feature of the apparatus is a very fine orifice at the spray nozzle through which insecticide is forced under pressure into a mixing chamber where it is further broken up by a blast of air under pressure coming from all sides. The air and the atomized insecticide then pass out through a somewhat larger opening in the cap of the spray nozzle. The spray produced is a mist and for practical purposes is not deposited on any surfaces. The cap of the nozzle can be adjusted closer to, or farther away from, the fine orifice, and with a lock nut can be set at any point of adjustment. The cap can be removed entirely for cleaning and the piece containing the fine orifice can also be readily removed. Should the small opening be stopped by a bit of dirt such as even filtering might not remove, it can be quickly cleared by removing this piece and reversing it in the stream of air under pressure.

The next essential feature is the bleeder connected with the air valve, which is opened when the air valve is closed. This bleeder is a small tube from the top of the insecticide reservoir to the outside through the air-valve mechanism. When the air valve is closed, air pressure on the insecticide is relieved through the bleeder tube. This prevents dripping at the nozzle.

The third essential feature is the use of a glass container for the insecticide set in a protective metal cover, through which have been cut windows so that the level of the insecticide may be observed. On the edge of the windows are calibrations from which the amount of insecticide contained in the reservoir can be read at a glance. The protective cover is constructed of a cylinder of dural metal open at the bottom and conical at the top, where it is threaded to fit into the sprayer. It is of such size that a glass cylinder will fit snugly inside of it. At the bottom a threaded disc with a gasket is screwed down on the lower rim of the glass cylinder. The glass cylinder is of a standard size and may be readily replaced if broken. The reservoirs at present in use hold 50 cc., but larger ones may be constructed, and probably will be, for use on the larger aircraft now being developed.

The air valve is of the push type, conveniently mounted, and is operated by the thumb of the person using the sprayer. A toggle valve will probably be used on larger sprayers. Air under pressure is

brought to the sprayer by a hose that fits in at the bottom of the handle. The best attachment is by means of a "Kwick-air" valve, which can be attached or removed by one-eighth turn of a milled sleeve.

The sprayer may be operated by compressed air at pressures from 25 to 50 pounds, but is most efficient when pressures between 30 and 40 pounds are used.

Figure 1 is a diagrammatic drawing, with explanatory notes, of the construction of the sprayer, figure 2 is a photograph of the finished apparatus, and figure 3 shows the sprayer in use.

STUDIES IN CHILDBIRTH MORTALITY

I. PUERPERAL FATALITY AND LOSS OF OFFSPRING ¹

By J. YERUSHALMY, *Statistician, United States Public Health Service*, M. KRAMER, *Assistant Statistician*, and E. M. GARDINER, *Director, Division of Maternity, Infancy, and Child Hygiene, New York State Department of Health*

Maternal deaths have rarely been studied in conjunction with all the births from which they arise. For example, the most extensive and valuable investigations in this country, the New York City study (1), and the Children's Bureau study in 15 States (2), are limited for the most part to an analysis of maternal deaths only. The distribution of maternal deaths according to the factors under investigation, when a similar distribution for the surviving mothers is not known, does not afford a measure of the risk of death associated with these factors. In fact, such evaluation is lacking not only for specific factors but also for the total risk of death due to childbearing. The maternal mortality rate, as ordinarily defined, is not strictly a measure of this risk; for, in the population exposed to risk, it neglects all pregnancies terminating in abortion or miscarriage, while the maternal deaths from these conditions are included. Even more restricted is our knowledge concerning the hazard to the mother associated with specific conditions such as the number of her previous pregnancies, her age, outcome of pregnancy (live birth, stillbirth, neonatal death), premature birth, and so on. If, in addition to the maternal deaths, the population exposed to risk were known in each case, the probability of death associated with such factors could be obtained. One valuable source from which information of this kind may be extracted is the birth certificate and the maternal death certificate. By themselves, these records are of limited value for study

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The authors are indebted to Miss S. Elizabeth Sheerar of the New York State Department of Health for her assistance in the preparation of the tables, and to Dr. Carroll E. Palmer of the U. S. Public Health Service and Dr. Allan F. Guttmacher of Johns Hopkins University for reading the manuscript and offering valuable suggestions.

purposes since the death certificate contains very little information concerning the birth. However, when the maternal death certificate is matched with the infant's birth certificate, a considerable amount of additional data becomes available. The same information is obtainable also for the births in which the mother survived and, thus, correlated investigation may be undertaken. This process of matching birth and death certificates was used in studying various phases of the problem of neonatal mortality and stillbirths (3, 4, 5, 6), and it was shown by various tests (4) that such errors as might have been entered on the birth certificate are not selective for the problems under investigation and that they are not of sufficient magnitude to affect the results seriously. The same method will, therefore, be used to investigate certain aspects of maternal mortality.

The procedure of matching birth and maternal death certificates is limited in that only the risk of death associated with the delivery of a viable offspring² can thus be studied. The fatality associated with abortions, miscarriages, ectopic pregnancies, and that of women who die undelivered cannot be studied by this method since no birth certificate is filed for these conditions. In order to avoid confusion with the term maternal mortality, the deaths of mothers who were delivered either of a live or stillbirth (per 10,000 total deliveries, including those of stillbirths) will be referred to as "puerperal fatality rate."³ This expression, puerperal fatality, appears to have certain advantages. It has the same meaning as the usual "case fatality rate," in the sense that it measures the mortality among a group of people all of whom are exposed to a given condition. It also parallels the meaning generally assigned to a case fatality rate in that it is restricted only to the length of time in which the condition exists (the postpartum period) whereas a mortality rate implies a calendar period of time (usually 1 year).⁴ It should be noted that the puerperal fatality rate is more nearly a measure of the risk of death associated with the delivery of a viable offspring, and should be a useful index in connection with the maternal health problem.

The investigation of various aspects of the problem of puerperal fatality will be presented in a series of papers of which this is the first. In these papers an attempt will be made to study puerperal fatality in its relation to such factors as outcome of pregnancy (live birth, stillbirth, neonatal death), premature birth, sex of infant, order of birth, age of mother, mother's previous infant losses, and so

¹ The term "viable offspring" will be used in this paper to denote a fetus which advanced at least to the fifth month of utero-gestation born alive or dead.

² The term "puerperal fatality" was suggested by Dr. Carroll E. Palmer of the U. S. Public Health Service.

³ Likewise, the ratio of maternal deaths due to abortion, miscarriage, and the like, to all women in the pregnant state may be termed "pregnancy fatality rate," and the ratio of all maternal deaths to all pregnant women may be designated as "maternal fatality rate."

on. For comparison, puerperal fatality rates will be paralleled in most cases with the stillbirth and neonatal mortality rates.

SCOPE OF THE STUDY

This series of studies is based on over a quarter of a million deliveries, and nearly 700 deaths which resulted from these deliveries, occurring in New York State (exclusive of New York City) in the 3-year period 1936-38. The information was obtained currently from birth and death certificates received by the New York State Department of Health. The names of all women who died from a puerperal cause were searched in the index of births to determine whether they were delivered of a live birth or of a stillbirth. When a birth certificate could not be found, additional searches were made to ascertain that no birth occurred. In most of these cases it was possible to determine from the statement on the death certificate the outcome of pregnancy, that is, whether pregnancy terminated in an abortion or miscarriage, whether it was ectopic, or whether the woman died undelivered. Similar searches were made in the file of birth certificates to ascertain the names of all infants who died under 1 month of age. The information from each of the matched birth and death certificates was brought together on a single punch card.

Only births and deaths occurring in New York State (exclusive of New York City) to resident mothers are included, since deaths of nonresidents which occurred after the mother and infant returned to their usual place of residence would not be found in the records of the State Department of Health. Since all births in the 3-year period 1936-38 form the basis of the study, only the deaths of mothers and infants associated with deliveries during this period are included. Deaths of mothers and infants occurring in 1936 but arising out of births in 1935 are excluded, and death of mothers and infants occurring in 1939 arising from births in 1938 are included.

THE DISTRIBUTION OF MATERNAL DEATHS BY OUTCOME OF PREGNANCY

During the 3-year period 1936-38, a total of 255,727 resident mothers of New York State were delivered of 258,525 live and still births.⁵ In the same period there occurred 1,122 deaths of women in which the primary cause of death was recorded as puerperal. The maternal mortality rate was therefore 43.4 per 10,000 total births (including stillbirths). A thorough search of the vital statistics files produced birth and stillbirth certificates for 689 deliveries in which death of the mother occurred, or 61.4 percent of the 1,122 maternal deaths. Of the remaining 433 deaths for which no birth certificate could be found, it was possible to establish with reasonable certainty,

⁵ There were 2,754 pairs of twins and 22 sets of triplets.

from the statements on the death certificate, that 224 (20.0 percent of the maternal deaths) were associated with abortion or miscarriage, 74 (6.6 percent) were ectopic pregnancies, and 93 (8.3 percent) of the mothers died undelivered. There were 24 additional women who probably died undelivered. In the remaining 18 cases, it was questionable whether death was associated with the delivery of a viable offspring or with an abortion or miscarriage. Since no birth or stillbirth certificate could be found in the New York State files, it is possible that a certain number of these women were delivered of a viable offspring in New York City or out of the State. It is possible also that some of these represent unregistered births.

It may be of interest to utilize the maternal deaths associated with abortion for the purpose of testing the estimates of the frequency of abortion and of the puerperal fatality associated with this condition. Taussig (7) proposes the following as estimates: The ratio of abortions (spontaneous and induced) to confinements 1-2.5 in urban localities and 1-5 in the rural districts; the mortality associated with abortions, 1.2 percent. Taussig also assumes that there are as many deaths from abortion which are concealed under nonpuerperal causes as there are registered deaths from this condition. Following Taussig's first assumption, the number of abortions which occurred in upstate New York would be as follows:

Locality	Confinements	Ratio of abortions to confinements	Estimated number of abortions
Urban.....	129, 898	1-2.5	51, 959
Rural.....	125, 829	1-5	25, 165
Total.....	255, 727	-----	77, 124

The total number of abortions in the 3-year period would therefore be 77,124. If Taussig's 1.2 percent fatality rate for abortions is accepted, the expected number of maternal deaths from this cause would be $77,124 \times .012$, a total of 925. The actual number accounted for was 224, with the possibility that all the 18 questionable cases were also due to abortion. When these 18 cases are added, a total of 242 registered deaths associated with this condition is obtained. If the assumption that there were an equal number of concealed deaths from abortion is accepted, there would be 484 deaths from abortion, whereas the estimated number is 925. For New York State, exclusive of New York City, it would appear either that abortions may not be as frequent as the above estimates indicate or that the mortality from abortions is lower than is assumed.

PUERPERAL FATALITY

For the study of puerperal fatality, maternal deaths associated with abortion, miscarriage, ectopic pregnancy, and women who die undelivered are excluded. There remain the 689 deaths which occurred among the 255,727 mothers who were delivered either of a live birth or a stillbirth. This paper, therefore, deals primarily with puerperal fatality in the sense described above, that is, with the risk of death to the mother who is delivered either of a live or a stillbirth. The criterion for distinguishing a stillbirth from a miscarriage is that the former has been registered as a stillbirth. Such registration, according to New York State law, is required if the fetus "advanced to the fifth month of utero-gestation."

The puerperal fatality rate in the 3-year period was 26.9 per 10,000 confinements.

TABLE 1.—Distribution of deaths of mothers of viable offspring by primary and secondary causes of death—New York State (exclusive of New York City), 1936-38

Secondary causes of death	International list numbers	Primary causes of death (International List Numbers, ¹ 1929 revision)										Total
		141 ²	144 (a)	144 (b)	145	146	147	148	149 (a)	149 (b)	150	
Puerperal causes (total).....	141-150	15	26	46	85	54	20	3	16	15	---	289
Abortion without septic condition ²	141 ²	---	5	---	1	21	4	2	---	2	---	35
Placenta praevia.....	144 (a)	---	---	7	---	---	---	---	---	---	---	7
Other hemorrhages.....	144 (b)	8	---	12	---	---	---	---	---	8	---	28
Puerperal septicemia.....	145	---	---	---	---	---	---	---	---	---	---	---
Puerperal albuminuria and eclampsia.....	146	1	2	6	10	---	---	---	---	---	---	19
Other toxemias.....	147	---	1	4	3	5	---	---	---	1	---	14
Puerperal embolism and thrombosis.....	148	---	3	4	13	7	3	1	16	3	---	50
Cesarean operation.....	149 (a)	6	13	11	33	19	9	---	---	1	---	92
Other accidents of childbirth.....	149 (b)	---	2	21	5	2	3	---	---	---	---	33
Other and unspecified.....	150	---	---	---	1	---	1	---	---	---	---	2
Nonpuerperal causes (total).....		25	3	18	46	32	13	19	34	60	1	251
Diseases of the heart.....	90-95	4	2	7	9	13	5	10	12	14	---	76
Pneumonia (all forms).....	107-109	5	---	3	7	6	2	1	5	17	1	47
Diseases of the digestive system.....	115-129	1	1	---	5	3	1	---	12	7	---	30
Cerebral hemorrhage.....	82	1	---	---	2	4	1	---	7	7	---	15
Chronic nephritis.....	131	7	---	2	---	---	1	2	---	---	---	12
Diseases of the female genital organs.....	139	---	---	---	7	---	---	---	---	---	---	7
Anemia.....	71	3	---	---	---	---	1	1	---	---	---	7
Other.....	4	---	6	17	8	---	5	3	14	---	---	57
Total secondary causes.....		40	29	64	131	86	33	22	50	75	1	531
No secondary cause.....		---	9	35	26	33	6	31	5	12	1	158
Total.....		40	38	99	157	119	39	53	55	87	2	689

¹ For convenience of printing, only the International List Numbers are given. The causes which these numbers represent are given under "secondary causes."

² Under this classification come also accidents of pregnancy of mothers of viable offspring.

Causes of puerperal fatality.—Table 1 gives the distribution of the 689 maternal deaths according to the primary and secondary causes of death as given on the death certificate and as classified by the Division of Vital Statistics according to the "Manual of Joint Causes of Death" (1929 Revision). It should be noted that the selection, by means of set rules, of a primary cause of death, when several causes

are stated on the death certificate, results in a classification of primary causes of death which is somewhat different from that of hospital statistics (8, 9), or that obtained from personal interview with the physician (2). The primary causes fall into four main groups: Toxemias of pregnancy (158 deaths), puerperal septicemia (157 deaths), accidents of childbirth (142 deaths), and puerperal hemorrhage (137 deaths). Accidents of pregnancy⁶ (40 deaths) and puerperal embolism and thrombosis (53 deaths) are responsible for the remaining deaths.

A somewhat different picture of the causes of maternal deaths is obtained when the secondary causes are considered. The distribution of the mothers dying *from* the various puerperal causes is different from that of the mothers dying *with* these causes. The addition of the frequencies with which a certain cause appears both as primary and as secondary cause of death gives the total frequency with which that cause occurred among the 689 deaths. The relative standing of the 4 main groups of causes in terms of total frequency and as primary causes appears as follows:

Group of causes	Number of times appearing as primary cause	Total number of times appearing on certificate
Toxemias of pregnancy.....	158	191
Puerperal septicemia.....	157	157
Accidents of childbirth.....	142	267
Puerperal hemorrhage.....	137	172

Thus, in terms of total frequency, accidents of childbirth were in first place, while as a primary cause of death they were in third place. Septicemia, which was the second largest in the primary group, was the smallest group in terms of total frequency. The most striking change occurred in cesarean operation which appeared on a total of 147 certificates but was coded as a primary cause of death in only 55 cases. The total frequency of puerperal septicemia was the same as the frequency with which it appeared as a primary cause. This is due to the fact that according to the "Manual of Joint Causes of Death" it takes preference over any other puerperal cause. Consequently, it never appeared as a secondary cause.

A secondary cause was given on 531, or 77.1 percent, of the maternal death certificates. Of these more than one-half (280) were again puerperal causes. The most common nonpuerperal causes were diseases of the heart of which there were 76, and pneumonia which appeared on 47 certificates. Among the puerperal causes, cesarean operation appeared most frequently as a secondary cause of death (92

⁶ This comes under International List number 141. The title "abortion without septic condition" is, in a sense, misleading since under this classification are coded also deaths of mothers of viable offspring when the cause of death is an accident of pregnancy.

deaths), followed by puerperal embolism and thrombosis which were given on 50 death certificates.

Secondary causes, largely nonpuerperal, were given in conjunction with accidents of childbirth more often than with any other cause. A nonpuerperal secondary cause was given in conjunction with accidents of childbirth more than twice as often as with the group of toxemias or with septicemia, and four times as frequently as in connection with the group classified as hemorrhage.

Interval between birth of child and death of mother.—From the statements on the birth certificate as to the date and time of birth and on the death certificate as to the date and time of death of the mother, it is possible to determine the length of time that elapsed between birth and maternal death.⁷ Table 2 gives the distribution of the maternal deaths by interval between birth of child and death of mother. The distribution of the deaths during the first month by days and that of the deaths under 1 day by hours is shown in figure 1.

TABLE 2.—*Distribution of deaths of mothers of viable offspring by the interval between birth of child and death of mother—New York State (exclusive of New York City), 1936-38*

Interval between birth of child and death of mother	Deaths		Maternal deaths within 24 hours of birth of child				
	Number	Percent	Hours	Number of deaths	Percent of deaths under 1 day	Percent of total deaths	
Under 1 day.....	257	37.3	{ Prior to birth Under 1 hour }	24	9.3	3.5	
1 day.....	40	5.8		39	15.2	5.7	
2 days.....	36	5.2		1	28	10.9	4.1
3 days.....	37	5.4		2	29	11.3	4.2
4 days.....	32	4.6		3	29	11.3	4.2
5 days.....	32	4.6		4	17	6.6	2.5
6 days.....	29	4.2		5	8	3.1	1.2
7 days.....	26	3.8		6	15	5.8	2.2
8 days.....	17	2.5		7	13	5.1	1.9
9 days.....	12	1.7		8	7	2.7	1.0
10 days.....	13	1.9		9	8	3.1	1.2
11 days.....	13	1.9		10	1	0.4	0.1
12 days.....	13	1.9		11	3	1.2	0.4
13 days.....	11	1.6		12	8	3.1	1.2
2 weeks.....	34	4.9	13-16	12	4.7	1.7	
3 weeks.....	26	3.8	17-20	9	3.5	1.3	
1 month.....	44	6.4	21-23	5	1.9	0.7	
2 months.....	17	2.5	Not stated	2	0.8	0.3	
Total.....	689	100.0	Total	257	100.0	37.3	
Under 1 week.....	463	67.2	0-6	159	73.5	27.4	
Under 1 month.....	628	91.1	0-12	229	89.1	33.2	

The average interval of time between birth of child and death of mother was 9.1 days. The first 24 hours after birth were the most fatal; 37 percent of all the deaths occurred within that interval. The mortality remained nearly constant for the next 3 days. There was a slight drop in the last 3 days of the first week and a sharper drop

⁷ For the distribution of total maternal mortality by interval, see (2).

thereafter. Two out of every three deaths occurred within the first week after birth. Similarly, the first hour took the greatest toll of deaths under 1 day. The mortality was fairly constant for the next 3 hours and decreased markedly thereafter. In the first 12 hours occurred 89 percent of all the deaths under 1 day and a third of all maternal deaths. The rate of mortality in the second 12 hours of the first day remained the same for the following 3 days.

There were 24 women who, according to the statements of hour on the two certificates, appeared to have died prior to the birth of the infant. In the majority of these cases the interval was of only a few minutes' duration. In most of these cases the child was born dead. The longest period between death of mother and birth of infant was 55 minutes.

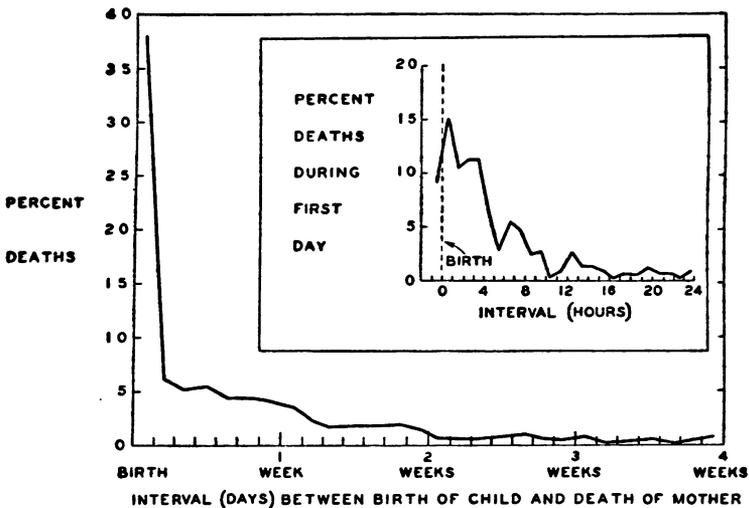


FIGURE 1.—Percentage distribution of puerperal deaths by interval between birth of child and death of mother. Deaths occurring in less than 1 month by daily interval and deaths occurring in less than 1 day by hourly interval, New York State, exclusive of New York City, 1936-38.

There were considerable differences in the average interval for the various causes of death, as may be seen from table 3 which gives the distribution of the deaths by cause and by interval between birth of child and death of mother.

Mothers dying from hemorrhage survived the shortest interval (2.2 days) and 73 percent of them died in less than 1 day. Mothers dying from septicemia had the longest interval (18.6 days), and only 2.5 percent of them died within 24 hours. The distribution by interval of septicemia deaths was different from the distribution of deaths from any other cause. These deaths were most numerous in the last 3 days of the first week after delivery. The average interval for mothers dying from toxemia was the same as that for mothers dying from accidents of childbirth (6.5 days).

TABLE 3.—Distribution of puerperal deaths by cause of death and by interval between birth of child and death of mother—New York State (exclusive of New York City), 1936-38

[Primary cause of death]

Interval between birth of child and death of mother	Total	Accidents of pregnancy, 141	Hemorrhage			Puerperal septicemia 145, 140, 142a	Toxemia			Puerperal phlegmasia, etc., 148	Cesarian, 149a	Other accidents of childbirth, 149b	Unspecified conditions, 150
			144a	144b	Total		146	147	Total				
NUMBER													
Under 1 day...	257	20	22	78	100	4	52	17	69	13	9	42	-----
Under 1 hour...	63	6	6	7	13	1	14	6	20	3	3	17	-----
1 day.....	40	3	3	4	7	2	17	2	19	1	3	5	-----
2 days.....	36	4	1	4	5	3	9	3	12	4	6	2	-----
3 days.....	37	4	1	3	4	6	7	4	11	1	7	4	-----
4 days.....	32	3	-----	2	2	13	3	-----	3	-----	6	5	-----
5 days.....	32	1	1	1	2	12	5	1	6	1	5	5	-----
6 days.....	29	1	3	2	5	12	2	1	3	-----	6	2	-----
1 week.....	105	2	5	4	9	42	10	10	20	15	6	11	-----
2-3 weeks.....	60	-----	2	1	3	30	5	1	6	9	4	7	-----
4 weeks and over.....	61	2	-----	-----	-----	33	9	-----	9	9	3	4	-----
Total.....	689	40	38	99	137	157	119	39	158	53	55	87	2
PERCENT													
Under 1 day...	37.3	50.0	57.9	78.8	73.0	2.5	43.6	43.6	43.7	24.5	16.4	48.3	-----
Under 1 hour...	3.5	15.0	15.8	7.1	9.5	0.6	11.8	15.4	12.7	5.7	5.5	19.5	-----
1 day.....	5.8	7.5	7.9	4.0	5.1	1.3	14.3	5.1	12.0	1.9	5.4	5.7	-----
2 days.....	5.2	10.0	2.6	4.0	3.6	1.9	7.6	7.7	7.6	7.5	10.9	2.3	-----
3 days.....	5.4	10.0	2.6	3.0	2.9	3.8	5.9	10.3	7.0	1.9	12.7	4.6	-----
4 days.....	4.6	7.5	-----	2.0	1.5	8.3	2.5	-----	1.9	-----	10.9	5.7	-----
5 days.....	4.6	2.5	2.6	1.0	1.5	7.6	4.2	2.6	3.8	1.9	9.1	5.7	-----
6 days.....	4.3	2.5	7.9	2.0	3.6	7.6	1.7	2.6	1.9	-----	10.9	2.3	-----
1 week.....	15.2	5.0	13.2	4.0	6.6	26.7	8.4	25.6	12.7	28.3	10.9	12.6	-----
2-3 weeks.....	8.7	-----	5.3	1.0	2.2	19.1	4.2	2.6	3.8	17.0	7.3	8.0	-----
4 weeks and over.....	8.9	5.0	-----	-----	-----	21.0	7.6	-----	5.7	27.0	5.4	4.5	-----
Total.....	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	-----
Average interval (in days).....	9.1	4.2	3.7	1.6	2.2	18.6	7.2	4.4	6.5	15.2	8.1	6.5	-----

The largest proportion of mothers dying in less than 1 day died from hemorrhage. Toxemia of pregnancy was the most frequent cause when death occurred either in less than 1 hour or within 1 to 3 days after delivery, and from the fourth day on the largest number of deaths occurred from septicemia.

Puerperal fatality and survival of offspring.—Puerperal fatality is only one part of the casualties of childbirth. The other is that of the infant in the form of stillbirth or neonatal mortality.⁸ The rate for each of these is over ten times as high as puerperal fatality. In many respects, the mortality of the mother is associated with that of the infant. The stillbirth and neonatal mortality rates of infants whose mothers die in childbirth is very much higher than those of infants whose mothers survive the postpartum period. Similarly, puerperal fatality increases sharply when the infant dies at birth or shortly thereafter.

⁸ In this paper, neonatal mortality is used to indicate mortality of infants under 1 month of age.

Of the 258,525 infants in the study, 7,177 were stillbirths and 7,550 died neonatally. The stillbirth rate was 27.8 per 1,000 total births and the neonatal mortality rate was 30.0 per 1,000 live births. Thus, out of every 1,000 total births, 57 were either stillborn or did not live to be 1 month old. Among the 248,697 mothers who were delivered of live births, there occurred 479 deaths, and of the 7,030 mothers of stillbirths, 210 died. The puerperal fatality rate was 19.3 per 10,000 deliveries when the infant was born alive and 298.7 when the infant was born dead. The rate was 16.6 for mothers whose infants survived the neonatal period and 108.0 for mothers of infants who died neonatally. Similarly, the rate for combined loss⁹ for infants of surviving¹⁰ mothers was 56.0 per 1,000 total births, while for the infants of the 689 maternal deaths it was 417.4. The excess in the rate was more pronounced in the case of stillbirths than it was in the case of neonatal mortality. The respective rates for infants of surviving mothers and for those of mothers who died were 27.0 and 300.4 for stillbirths, and 29.8 and 167.3 for neonatal mortality. The increase in puerperal fatality associated with infant loss was greater than that in the rate for infant loss associated with death of mother. The ratio of puerperal fatality among mothers whose infants were lost to that of mothers of surviving infants was 12.2, while the ratio of the rate for combined infant loss⁹ when the mother died in childbirth to that of infants whose mothers survived was 7.5. The ratios were 11.1 for stillbirth and 5.6 for neonatal mortality. These facts are brought out in figure 2 and table 4, which is a fourfold table presenting the outcome of pregnancy for mother and infant. From this table, it is possible to determine the probabilities of losing through puerperal fatality, stillbirths, and neonatal mortality mother only, infant only, and both mother and infant. In terms of chances per 10,000 deliveries these probabilities were:

	<i>Chances per 10,000 deliveries</i>
Losing infant only.....	558. 2
Losing mother only.....	15. 7
Losing both mother and infant.....	11. 3

The increase in puerperal fatality associated with loss of infant was present to a considerable degree in all causes of death, as may be seen from table 5. There were, however, notable differences in the relative frequencies of the various causes by outcome of pregnancy. Septicemia was the most common cause of death in the mothers who were delivered of live births, while toxemias of pregnancy were the largest group for the mothers of stillbirths. Accidents of childbirth were second in importance as a cause of death for mothers of live

⁹ The term "combined loss" denotes the number of stillbirths and neonatal deaths combined per 1,000 total births.

¹⁰ Mothers who did not die from a puerperal cause.

births, while puerperal hemorrhage was the second largest group for the mothers of the stillborn infants. The least frequent cause when the infant was born dead was cesarean operation, and the ratio of the mortality of mothers of stillbirths to that of mothers of live births was least for this cause. Similarly, septicemia and accidents of child-birth were, respectively, the first and second causes when the infant survived the neonatal period, while toxemia and septicemia had the corresponding respective roles for the mothers of the neonatal deaths.

TABLE 4.—*Distribution of births according to survival of mother and infant—New York State (exclusive of New York City), 1936-38*

Mother	Infant				Total	Rates for infant loss		
	Survived	Died				Combined loss ¹	Neonatal mortality ²	Still-birth ³
		Total	Neonatal death	Still-birth				
Survived.....	243,385	14,431	7,467	6,964	257,816	56.0	29.8	27.0
Died.....	413	296	83	213	709	417.4	167.3	300.4
Total.....	243,798	14,727	7,550	7,177	258,525	57.0	30.0	27.8
Puerperal fatality ⁴	16.6	202.8	108.0	298.7	26.9			

¹ Per 1,000 total births (including stillbirths).
² Per 1,000 total live births.
³ Per 1,000 total births (including stillbirths).
⁴ Per 10,000 deliveries.

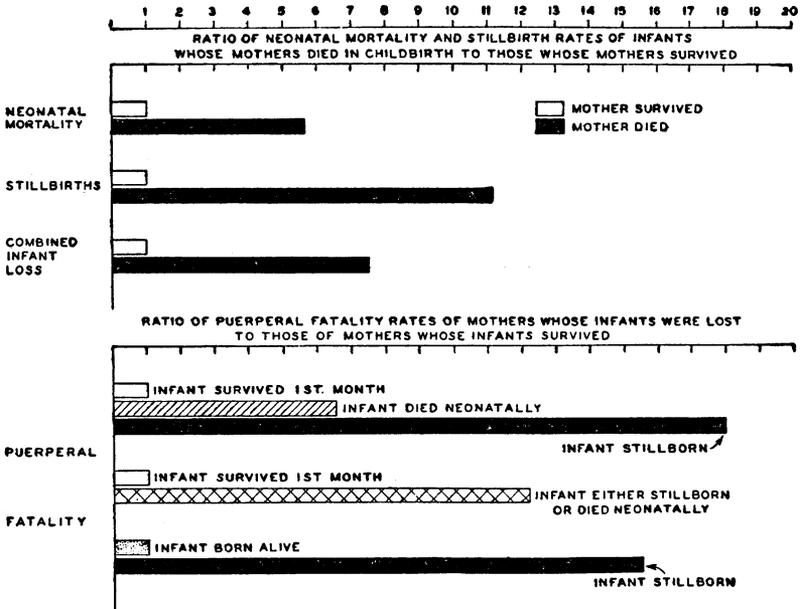


FIGURE 2.—Puerperal fatality in relation to survival of offspring and infant loss in relation to survival of mother, New York State, exclusive of New York City, 1936-38.

Of the infants whose mothers died, the largest proportion of survivors resulted from mothers who died from cesarean operation and the least number from those who died from accidents of pregnancy. Only 43.0 percent of the infants of mothers who died from toxemia survived the neonatal period. The corresponding percentage for the infants of the hemorrhage group was 56.2; for infants of mothers dying from septicemia the percentage was 68.8; and 73.9 percent of the infants whose mothers died from accidents of childbirth lived to be 1 month old.

TABLE 5.—*Puerperal fatality rates by cause of death and by survival of offspring—New York State (exclusive of New York City), 1936-38*

Cause of death	Total	Offspring				Total	Offspring					
		Live birth			Still-birth		Live birth				Still-birth	Com-bined loss
		Total	Sur-vivor	Neo-natal death			Total	Sur-vivor	Neo-natal death			
		Number of maternal deaths					Puerperal fatality rates ¹					
Accidents of pregnancy.....	40	14	6	8	26	15.6	5.6	2.5	10.8	369.8	238.5	
Puerperal hemorrhage.....	137	87	77	10	50	53.6	35.0	31.9	138.4	711.2	421.0	
(a) Placenta praevia.....	38	20	16	4	18	14.9	8.0	6.6	55.4	256.0	154.4	
(b) Other puerperal hemorrhages.....	99	67	61	6	32	38.7	26.9	25.3	83.1	455.2	266.6	
Puerperal septicemia and pyemia (not specified as due to abortion).....	157	131	108	23	26	61.4	52.7	44.7	318.4	369.8	343.8	
Toxemias of pregnancy.....	158	92	68	24	66	61.8	37.0	28.2	332.3	938.8	631.4	
Puerperal albuminuria and eclampsia.....	119	70	55	15	49	46.5	27.8	22.8	207.7	697.0	449.0	
Other toxemias of pregnancy.....	39	22	13	9	17	15.3	8.8	5.4	124.6	241.8	182.4	
Puerperal embolism and thrombosis (not specified as septic).....	53	43	36	7	10	20.7	17.3	14.9	96.9	142.2	119.3	
Other accidents of childbirth.....	142	110	105	5	32	55.5	44.2	43.5	69.2	455.2	259.6	
(a) Cesarean operation.....	55	49	47	2	6	21.5	19.7	19.5	27.7	85.3	56.1	
(b) Others.....	87	61	58	3	26	34.0	24.5	24.0	41.5	369.8	203.5	
Other and unspecified conditions of the puerperal state.....	2	2	1	1	-----	0.8	0.8	0.4	13.8	-----	7.0	
All puerperal causes.....	689	479	401	78	210	269.4	192.6	166.1	1,078.9	2,987.2	2,020.6	

¹ Number of puerperal deaths per 100,000 deliveries in each category from each specified cause.

Parallel to the variations of cause of death by survival of offspring, there were notable differences in the length of the interval between birth of child and death of mother. The interval was considerably shorter for the mothers of stillbirths than it was for the mothers of live births, the respective intervals being 4.9 and 10.9 days. More than one-half of the deaths of mothers of stillbirths occurred within 24 hours of delivery, while only a third of the deaths of the mothers of live births occurred in this interval. The difference in the interval for the deaths of mothers whose infants survived the neonatal period and that of mothers whose infants died neonatally was considerably smaller. The average interval for the former was 11.2 days and for the latter it was 9.3 days.

Premature delivery.—The puerperal fatality as related to the period of gestation cannot be given in detail from the material at hand since the birth certificates until 1938 did not record the month of gestation. The only statement made was whether the child was full term or premature. Furthermore, since the weight and length of the infant are not given on the birth certificate, the statement by the physician that the birth was premature had to be accepted. However, the frequency of premature birth as obtained from the birth certificate agrees well with other studies in which the weight of the infant was taken as a criterion of prematurity (10). Furthermore, this information was available for two counties in the State in which special surveys were made and the known weight of the infant compared well with the statement of prematurity on the birth certificate.

Of the 255,727 deliveries, 13,727 were stated to be premature. The frequency of premature deliveries was therefore 53.7 per 1,000 total deliveries. The stillbirth and neonatal mortality rates of prematurely born infants is, as is known, very high. Of the 14,562 infants resulting from the premature deliveries, 3,608 were stillbirths and 4,095 were neonatal deaths. The stillbirth rate for the premature infants was 247.8 per 1,000 total premature births and the neonatal mortality rate was 373.8 per 1,000 premature live births. Thus, more than one-half of the prematurely born infants did not live to be 1 month old. The puerperal fatality associated with premature delivery was also considerably higher than that associated with full term deliveries. However, the increase in fatality associated with premature delivery is not as great for the mother as it is for the infant. Of the 242,000 mothers who delivered full term infants, 487 died in childbirth, and of the 13,727 mothers whose pregnancy terminated prematurely, 194 died. The respective puerperal fatality rates per 10,000 deliveries in the two groups were 20.1 and 141.3.

Table 6 presents the puerperal fatality rates by cause of death for full-term and premature births. The most frequent causes of death of the mothers who were delivered at term were septicemia and accidents of childbirth, while toxemias of pregnancy were by far the most frequent for the mothers whose pregnancy terminated prematurely. The puerperal fatality from cesarean operation was the same in the two groups.

As a probable result of the difference in causes of death, the interval between delivery and death was much longer for the full term maternal deaths (10.0 days) than for those in the premature group (6.1 days).

The increase in the puerperal fatality rates of premature deliveries was more pronounced for mothers of live births than for mothers of stillbirths. The respective rates in the full term and premature groups were 16.5 and 74.9 for the mothers of live births, and 266.2

and 318.7 among the mothers of stillbirths. The puerperal fatality among the mothers of neonatal deaths was the same whether the delivery was full term or premature. For mothers of infants who survived the neonatal period, the puerperal fatality rates in the full term and premature groups were 15.3 and 56.9, respectively.

TABLE 6.—*Puerperal fatality rates by cause of death for full term and premature deliveries—New York State (exclusive of New York City), 1936-38*

Cause of death	Number of deaths			Puerperal fatality rates ¹	
	Full term	Premature	Term not stated	Full term	Premature
Accidents of pregnancy.....	4	36	-----	1.7	262.3
Puerperal hemorrhage.....	109	27	1	45.0	196.7
(a) Placenta praevia.....	24	14	-----	9.9	102.0
(b) Other puerperal hemorrhages.....	85	13	1	35.1	94.7
Puerperal septicemia and pyemia (not specified as due to abortion).....	128	25	4	52.9	182.1
Toxemias of pregnancy.....	75	82	1	31.0	597.4
(a) Puerperal albuminuria and eclampsia.....	57	62	-----	23.6	451.7
(b) Other toxemias of pregnancy.....	18	20	1	7.4	145.7
Puerperal embolism and thrombosis (not specified as septic).....	42	11	-----	17.4	80.1
Other accidents of childbirth.....	127	13	2	52.5	94.7
(a) Cesarean operation.....	51	3	1	21.1	21.9
(b) Others.....	76	10	1	31.4	72.8
Other and unspecified conditions of the puerperal state.....	2	-----	-----	0.8	-----
Total.....	487	194	8	201.2	1413.3

¹ Number of puerperal deaths per 100,000 deliveries in each category from each specified cause.

Sex of infant.—The excess mortality of male infants over that of females is manifested also at birth. The stillbirth and neonatal mortality rates are considerably higher for boys than for girls. Of the 133,251 boys in this study, 3,914 were stillbirths and 4,422 died under 1 month of age. Among the 125,274 girls, there were 3,263 stillbirths and 3,128 neonatal deaths. The respective rates for boys and girls were 29.4 and 26.0 for stillbirths, and 34.2 and 25.6 for neonatal mortality. A priori considerations may lead to the expectation that puerperal fatality is also higher among mothers of boys than among mothers of girls. Boys are generally bigger and weigh more at birth and hence more complications may be expected. Furthermore, due to the higher stillbirth and neonatal mortality rates among boys, there are relatively more deliveries associated with loss of offspring among mothers of boys than among mothers of girls. Because of the association between survival of mother and offspring, higher puerperal fatality rates among mothers of boys might be expected to result from this fact alone. Actually no difference was noted in the total puerperal fatality rate by sex of offspring. The excess in the number of deliveries associated with the loss of male offspring was compensated by a relatively lower puerperal fatality

rate among the mothers of boys who were lost than among the mothers of girls who were born dead or who died neonatally.

There were 349 deaths among the mothers of boys and 334 deaths among mothers of girls.¹¹ The puerperal fatality rate was 26.2 for mothers of boys and 26.7 for mothers of girls.¹² Puerperal fatality was lower for mothers of boys than for mothers of girls when the infant died at birth or during the first month. The rates were 176.3 for mothers of boys and 217.5 for mothers of girls.¹³ Among mothers whose infants survived the neonatal period, the fatality did not differ by sex of offspring. An increase in puerperal fatality was associated with female births both when the infant was stillborn and when it died during the first month. The respective rates for mothers of boys and girls were 275.9 and 306.5 in the case of stillbirths, and 86.2 and 124.7 in the case of neonatal deaths. Due to the relatively small number of deliveries available for study in each of these cases it is not possible to determine whether these differences are real or whether they arise from sampling variations. It would be of interest to investigate this point further on a larger sample of births.

Plural deliveries.—There were 2,776 plural deliveries during the 3-year period, resulting in 2,754 pairs of twins and 22 sets of triplets. The frequency of plural births was 10.9 per 1,000 deliveries. The total number of plural infants was 5,574. There were 21 deaths among the mothers of plural births, and the puerperal fatality rate for these mothers was 75.6, or nearly three times as high as the total puerperal fatality rate. This increase is of the same magnitude as the increase in total infant loss among plural births. The rate for combined loss among the plural births was 169.2. The stillbirth rate was 52.6 and the neonatal mortality rate was 123.1.

Of the 2,776 plural deliveries, 843 were of unlike-sexed and 1,933 were of like-sexed infants. In the first group there were 5 maternal deaths, and in the second group there were 16. The puerperal fatality rate was 59.3 for mothers of unlike-sexed and 82.8 for mothers of like-sexed plural births. Since the unlike-sexed twins are dizygotic while the like-sexed are a composite group consisting of both monozygotic and dizygotic, it is indicated that the puerperal fatality associated with the birth of monozygotic twins may be higher than that associated with the birth of dizygotic twins. The figures, however, are not large enough for safe conclusions.¹⁴

¹¹ In addition, 5 maternal deaths were delivered of twins of different sexes and the sex of the infant of another maternal death was not stated.

¹² These rates are based on births rather than on deliveries in order to avoid the complicating factor of unlike-sexed multiple deliveries. Rates based on births differ only very slightly from those based on deliveries since multiple births form about one percent of all deliveries.

¹³ The difference in these rates is on the border line of statistical significance, being slightly less than twice the standard deviation of the difference.

¹⁴ The difference between the rates is not statistically significant.

Deaths of unwed mothers.—There were 5,764 unwed mothers in the study who were delivered of 5,812 infants. The frequency of illegitimate deliveries was 22.5 per 1,000. There were 15 maternal deaths in this group and the puerperal fatality rate was 26.0. This is practically the same as the fatality of legitimate deliveries. The infants of these mothers, however, did not fare as well. The stillbirth rate for illegitimate infants¹⁵ was 60.0 and the neonatal mortality rate was 63.2. The rate for combined infant loss was 119.4. There may be an extra risk to the unwed mother which is hidden among the deaths associated with abortion. This, however, cannot be determined from the material at hand, since, as was stated previously, the population exposed to risk is not known. But it is of particular interest that among the unwed mothers in this study who were delivered of a viable infant, the puerperal fatality was not greater than that of the married mothers.

Residence of mother.—There were no differences in puerperal fatality between the mothers residing in urban localities and those living in the rural districts. The puerperal fatality rate was 26.9 for the former and 27.0 for the latter. Similarly, the chances of losing the infant were practically the same whether the mother resided in a city or in the country.

SUMMARY

This paper is the first of a series on puerperal fatality, which is defined as the risk of death to the mother associated with the delivery of a viable offspring. These studies will consider puerperal fatality in its relation to such factors as outcome of pregnancy (live birth, stillbirth, neonatal death), premature delivery, parity, age, sex of infant, number of previous infant losses, and so on. Puerperal fatality rates will be paralleled with the stillbirth and neonatal mortality rates.

The studies are based on over a quarter of a million deliveries and nearly 700 maternal deaths occurring in New York State (exclusive of New York City) in the 3-year period 1936–38. The information was obtained from routine vital statistics records of births and deaths.

The maternal death certificate was matched with the birth or stillbirth certificate of the infant. Similarly, the death certificate of infants who died under 1 month of age was matched with the birth certificate of the same infant. The information from the matched certificates was brought together on a single punch card.

This first paper records the following findings concerning puerperal fatality and loss of offspring:

1. The average interval between birth of child and death of mother was 9.1 days. The first 24 hours after delivery were the most fatal,

¹⁵ Recent legislation in New York State forbids the mention of illegitimacy on the birth certificate. In the last 2 years of the study a child was considered illegitimate if the father's name was omitted from the birth certificate.

accounting for 37 percent of all deaths. Similarly, the first hour after the birth of the child took the greatest toll of the deaths under 1 day. Mothers dying from hemorrhage survived the shortest average interval (2.2 days); mothers dying from septicemia, the longest interval (18.6 days).

2. There was a strong association between death of mother and loss of offspring. The puerperal fatality rate was 19.3 per 10,000 deliveries when the infant was born alive and 298.7 when the infant was born dead. The rate was 16.6 for mothers whose infants survived the neonatal period and 108.0 for mothers whose infants died under 1 month of age. Similarly, the stillbirth rate was 27.0 per 1,000 total births for infants of surviving mothers and 300.4 for infants whose mothers died in childbirth. The corresponding neonatal mortality rates were 29.8 and 167.3 per 1,000 live births. Septicemia was the most frequent cause of death for mothers of live births, while toxemia was the most frequent cause for the mothers of stillbirths. The average interval between delivery and death was 10.9 days for mothers whose infants were born alive and 4.9 days for mothers of stillborn infants.

3. Mortality of both mother and infant increased sharply when pregnancy terminated prematurely. This increase was more pronounced for infants than for mothers. The puerperal fatality rate was 20.1 when birth was at term and 141.3 when it was premature. The stillbirth rate for premature infants was 247.8 and the neonatal mortality rate was 373.8. Septicemia was the most common cause of death for the mothers who went to term, while toxemia was by far the most frequent cause of death for the mothers of the premature. The increase in the puerperal fatality rate of premature births was more marked when the infant survived the neonatal period than when the infant was lost.

4. No difference was noted in the puerperal fatality rate by sex of infant, while the stillbirth and neonatal mortality rates were considerably higher for boys than for girls. The respective rates for males and females were 29.4 and 26.0 for stillbirths and 34.2 and 25.6 for neonatal mortality.

5. The puerperal fatality rate, as well as the stillbirth and neonatal mortality rates, of plural births were three times as high as the rates for single births.

6. Unwed mothers who were delivered of viable offspring were exposed to fatality rates which were practically the same as those of married mothers. The infants of the unmarried mothers, however, had higher mortality rates than legitimate infants. The stillbirth rate for illegitimate infants was 60.0 and the neonatal mortality rate was 63.2, compared to the total respective rates of 27.8 and 30.0.

7. The puerperal fatality rate, as well as the stillbirth and neonatal mortality rates, was the same whether the mother resided in an urban locality or in a rural district.

8. Use was made of the maternal deaths associated with abortion for the purpose of testing estimates on the frequency of abortions and on the mortality associated with this condition. It appears that, at least for New York State exclusive of New York City, the estimates are too high.

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LEPROSY: VITAMIN B₁ DEFICIENCY AND RAT LEPROSY

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That there exists a relation between nutrition and leprosy has been believed by some investigators for many years and statements relative to such a relation have appeared in the literature with increasing frequency. Since we are unable to study human leprosy in laboratory animals, it is necessary to confine our nutritional studies to rat leprosy in the rat.

The results of some laboratory investigations of various nutritional deficiencies have been reported, of which but a few have dealt specifically with vitamin B₁ deficiencies while others have dealt with deficiencies in the vitamin B complex.

Muir and Henderson (1) in 1928 reported on a few studies of the relation of vitamin deficiency to rat leprosy and concluded that increased virulence of the infection does not occur in rats fed on vitamin deficient diets.

Lamb (2) in 1935 reported on his studies of the effects of malnutrition on the pathogenesis of rat leprosy, of which he made the following summary: "Subcutaneous inoculations of rat leproma in a large number of rats on many kinds of dietary deficiencies yielded generally negative results. * * * An exceptional case was a diet of starchy foods plus boiled taro root and fish, which repeatedly increased the development of subcutaneous lesions.

"Upon using intracardia inoculations of rat leproma, diets deficient in the vitamin B complex and somewhat low in protein produced an extensive increase in visceral lesions of rat leprosy * * *."

Badger and Sebrell (3) in 1935 in preliminary studies found that the incubation period of rat leprosy in white rats on a vitamin B₁ deficient diet was appreciably shorter than in the rats fed a well balanced diet.

Lampe and de Moor (4) in 1935 found that vitamin B₂ deficiency had some influence in aiding the development of rat leprosy after percutaneous inoculation with moderate doses.

Lampe et al. (5) in 1936, in their report on infection of rats through the shaven skin from infected mud, stated that vitamin B₁ and B₂ hypovitaminosis appears to be necessary to produce the infection.

From the results of the investigations here reported, it may be stated that—

1. The incubation period of rat leprosy is of shorter duration in vitamin B₁ deficient than in normal rats.
2. After becoming established in rats inoculated subcutaneously, the leproma at the site of inoculation develops more rapidly and becomes much larger in the normal than in the vitamin B₁ deficient rats.
3. Gross evidence of generalization of the infection appears much earlier in the vitamin B₁ deficient than in the normal control rats.

Incubation period.—The duration of the incubation period can be determined with any degree of accuracy only following subcutaneous inoculation and only when the palpable foreign tissue reaction has completely disappeared. Because the palpable foreign tissue reaction persists in some of the deficient rats until after the true leproma becomes palpable, even following small doses of a dilute inoculum, it has been impossible in the majority of our experiments to determine accurately the period of incubation. In those experiments where the incubation period could be determined, it was definitely shorter in the vitamin B₁ deficient than in normal rats as illustrated in table 1.

Development of the leproma at the site of inoculation.—After it has become established, the leproma at the site of subcutaneous inocula-

tion develops more rapidly and becomes much larger in the normal, well nourished than in the vitamin B₁ deficient rats.

TABLE 1.—Incubation period of rat leprosy following subcutaneous inoculation in vitamin B₁ deficient and normal rats. Percent of rats with palpable leproma

Experiment No.	Group of rats	Number of rats	Percent of rats with palpable lepromata, after inoculation							
			First week	Second week	Third week	Fourth week	Fifth week	Sixth week	Seventh week	Eighth week
II.....	Vitamin B ₁ deficient.....	25	0	40.0	68.0	100.0	-----	-----	-----	-----
	Control.....	25	0	4.0	24.0	56.0	96.0	96.0	96.0	100.0
VIII.....	Vitamin B ₁ deficient.....	24	0	0	8.3	29.1	50.0	87.5	95.8	100.0
	Control.....	24	0	0	4.1	8.3	33.3	79.1	95.8	100.0

To determine the size of the lepromata, each was measured in two dimensions. From these measurements the area involved and the average area of involvement in the rats of each group were determined. These measurements do not take into account the thickness of the lepromata which was much greater in the normal than in the deficient rats. A marked variation occurred in the size of the lepromata in the rats within the groups. After 6 to 8 months the lepromata in some of the deficient rats were larger in areas involved than in some of the control rats but they continued to remain thinner. Frequently in the deficient rats there was seen only a scar at the site of inoculation. More often they had the appearance of progressively spreading lesions, while those of the controls were more circumscribed until much later.

The differences in the size of the lepromata in the vitamin B₁ deficient and normal rats in some of the experiments are shown in table 2.

TABLE 2.—Size of lepromata at the site of subcutaneous inoculation in vitamin B₁ deficient and normal rats

Number of weeks after inoculation	Experiment No.	Group of rats	Number of rats	Measurements of lepromata in mm.	Area of lepromata in square mm.	
					Variation	Average size
8.....	XXII.....	Vitamin B ₁ deficient.....	28	2 x 2 to 10 x 17.....	4 to 170.....	42.1
		Control.....	28	10 x 15 to 25 x 30.....	150 to 750.....	453.6
	XXV.....	Vitamin B ₁ deficient.....	13	2 x 2 to 7 x 15.....	4 to 105.....	27.8
		Control.....	13	12 x 18 to 22 x 22.....	216 to 484.....	365.9
12.....	X.....	Vitamin B ₁ deficient.....	10	2 x 2 to 15 x 17.....	4 to 255.....	62.6
		Control.....	10	10 x 20 to 27 x 30.....	200 to 810.....	431.1
16.....	X.....	Vitamin B ₁ deficient.....	7	5 x 10 to 15 x 20.....	50 to 300.....	175.0
		Control.....	9	15 x 15 to 25 x 25.....	223 to 625.....	404.0
	XVI.....	Vitamin B ₁ deficient.....	10	3 x 10 to 13 x 19.....	30 to 247.....	113.5
		Control.....	10	8 x 20 to 25 x 35.....	160 to 875.....	893.6
16-17.....	XL.....	Vitamin B ₁ deficient.....	22	Scar to 17 x 17.....	0 to 289.....	116.6
		Control.....	22	7 x 10 to 23 x 30.....	70 to 690.....	314.6
20.....	XVI.....	Vitamin B ₁ deficient.....	8	3 x 7 to 12 x 20.....	21 to 240.....	118.7
		Control.....	8	20 x 25 to 30 x 40.....	500 to 1,200.....	773.0
32.....	XVIII.....	Vitamin B ₁ deficient.....	16	4 x 10 to 35 x 50.....	40 to 1,850.....	610.9
		Control.....	16	20 x 23 to 50 x 65.....	460 to 3,250.....	1,401.0

It is believed that the shorter incubation period and the smaller lepromata at the site of subcutaneous inoculation in the deficient rats were not due specifically to vitamin B₁ deficiency but to an interference with the cellular defense mechanism of the animal brought about by a poor state of nutrition.

The incubation period has been found to be in indirect proportion to the state of nutrition of the rat as signified by weight. In experiment II (table 1), 4 weeks after inoculation, 100 percent of the deficient rats had palpable lepromata while but 56 percent of the control rats had palpable lepromata. At that time each of the deficient rats weighed less than when placed on the diet and showed an average loss of 24.1 percent, while the control rats had gained an average of 185.2 percent. A group of rats deficient in vitamin B₂ was also studied in this experiment. Four weeks after inoculation 80 percent of this group of rats had palpable lepromata, and their average weight was 2 percent greater than when placed on the diet. In experiment VIII (table 1), 29.1 percent of the vitamin B₁ deficient rats, 4 weeks after inoculation, had palpable lepromata while but 8.3 percent of the control rats had palpable lepromata. At that time the average weight of the deficient rats was but 21.5 percent greater than when placed on the diet, while that of the control rats was 195.5 percent greater.

RELATION OF STATE OF NUTRITION TO THE SIZE OF THE LEPRUMATA IN RATS ON VITAMIN B₁ DEFICIENT, PARTIAL VITAMIN B₁ DEFICIENT AND NORMAL. EXAMINATION 12 WEEKS AFTER SUBCUTANEOUS INOCULATION. EXPERIMENT X

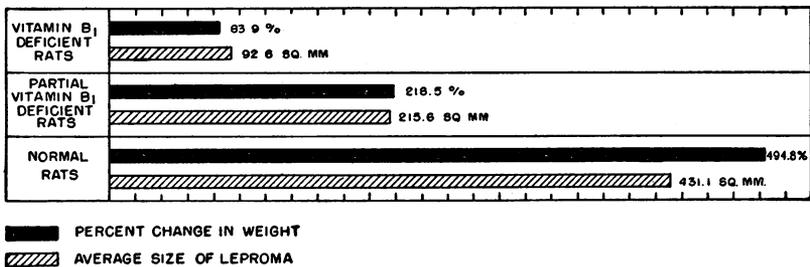


FIGURE 1.

The size of the leproma at the site of subcutaneous inoculation is in direct proportion to the state of nutrition of the rat as signified by the change in weight. This relation is well illustrated in the following two experiments and in figure 1.

In the first experiment vitamin B₁ deficient, partially vitamin B₁ deficient, and normal rats were studied. Twelve weeks after inoculation the average sizes of the lepromata of 10 rats of each group were 92.6, 215.6, and 431.1 sq. mm., respectively, while the average weights of the rats were 83.9, 281.5, and 494.8 percent greater than when placed on the diets.

In the second experiment rats malnourished by means other than vitamin deficiency were studied in addition to a vitamin B₁ deficient group. The state of malnutrition in one group was produced by limiting the food intake, in another by limiting the water intake, and in another by limiting the protein intake. Eight weeks after inoculation the average sizes of the lepromata of 13 rats of each group were: Vitamin B₁ deficient, 27.8 sq. mm.; low food intake, 79.9 sq. mm.; low water intake, 187 sq. mm.; low protein (4 percent casein), 241.6 sq. mm.; normal controls, 365.9 sq. mm. The average percentages of gain in weight of the rats examined were 3.7, 49.6, 66.6, 114.5, and 237.5, respectively (see figure 2).

RELATION OF STATE OF NUTRITION TO THE SIZE OF LEPROMATA IN RATS ON VITAMIN B₁ DEFICIENT, LOW FOOD INTAKE, LOW WATER INTAKE, LOW PROTEIN INTAKE AND CONTROL DIETS. EXAMINATION 8 WEEKS AFTER SUBCUTANEOUS INOCULATION.
EXPERIMENT XXV

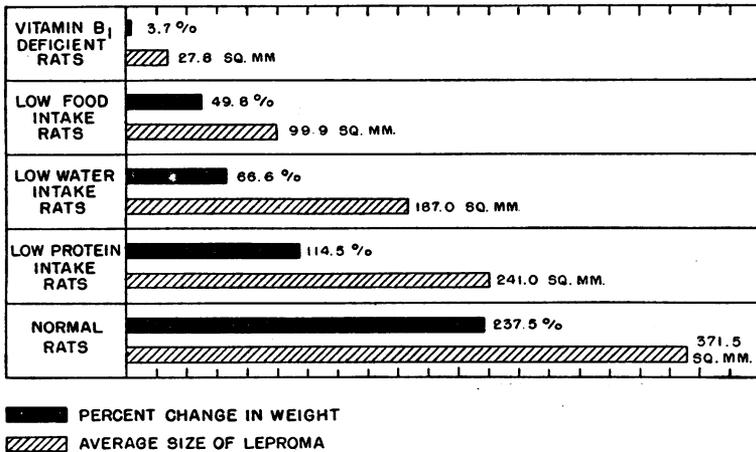


FIGURE 2.

The results obtained in several studies lend proof to the belief that these differences are due to an interference in the cellular defense mechanism in the poorly nourished animals.

The foreign-tissue reaction, resulting from subcutaneous inoculation, disappears more quickly in the well nourished than in the poorly nourished animals. Following subcutaneous inoculation a palpable foreign-tissue reaction may develop within 10 hours. The time of development of this reaction was determined in a group of rats. Six hours after inoculation none of the rats had palpable lesions, in 10 hours a few had such lesions, and after 24 hours this reaction had developed in most of the rats. The size of this palpable foreign-tissue reaction and the rate of its disappearance, usually within 7 to 10 days in normal rats, are somewhat dependent on the concentration of the inoculum. The rate of disappearance of the palpable foreign-

tissue reaction in normal and malnourished rats is illustrated in table 3.

TABLE 3.—Disappearance of the foreign tissue reaction following subcutaneous inoculation of leprosy material into vitamin B₁ deficient and well nourished rats; percent of rats with palpable reactions

Experiment No.	Group of rats	Number of rats	Percent of rats with palpable reactions, after inoculation									
			First day	Second day	Third day	Fourth day	Fifth day	Sixth day	Seventh day	Eighth day	Ninth day	Tenth day
VI.....	Vitamin B ₁ deficient.....	48				95.8		95.8		89.5		83.3
	Control.....	48				100.0		93.7		50.0		27.0
VIII.....	Vitamin B ₁ deficient.....	24			87.5		50.0		28.1			8.3
	Control.....	24			87.5		45.8		20.8			0
X.....	Vitamin B ₁ deficient.....	24		100.0			100.0		86.2			
	Control.....	24		100.0			95.8		66.6			
XVIII.....	Vitamin B ₁ deficient.....	46		95.5		88.8			62.2			
	Control.....	46		73.9		50.0			15.2			
XXII.....	Vitamin B ₁ deficient.....	40	95.0		90.0		52.5		15.0			
	Control.....	40	87.5		50.0		17.5		12.5			

To determine whether the difference in the rate of disappearance of the foreign-tissue reaction in poorly nourished and normal rats occurred only following inoculation of leprosy tissue, in two experiments vitamin B₁ deficient and normal rats were inoculated subcutaneously with both normal subcutaneous and leprosy tissue, one under the left and the other under the right ventral surface. The foreign tissue reactions at the sites of both inoculations disappeared more quickly in the normal than in the malnourished rats.

An experiment was conducted to determine whether, by stimulating the cellular response to inoculation, the foreign-tissue reaction would disappear more rapidly and the lepromata develop more slowly. In the belief that an intravenous inoculation would produce a marked cellular response, a group of 29 rats was inoculated both intravenously and subcutaneously and another group of 29 subcutaneously only, with an emulsion of a rat leproma. The palpable foreign-tissue reaction disappeared much more quickly in the rats which received both the intravenous and subcutaneous inoculations than in the rats which received only the subcutaneous inoculation. The incubation period was much longer in the rats which received both the intravenous and subcutaneous inoculations than in the rats which received only the subcutaneous inoculation. The results of this experiment are shown in table 4.

To learn to what extent intravenous inoculation of leprosy material produced a cellular response, a group of rats was so inoculated, and the change in the number of circulating white blood cells was

determined in a few. The average total numbers of white blood cells in the circulating blood of the rats studied were as follows: Previous to inoculation, 3,937; day after inoculation, 8,275; 2 days, 11,062; 3 days, 13,650; 5 days, 14,150; and 7 days, 16,200. After the seventh day the number gradually decreased.

TABLE 4.—*Disappearance of palpable foreign tissue reactions and the appearance of palpable lepromata in rats inoculated both subcutaneously and intravenously and in rats inoculated subcutaneously only*

Disappearance of foreign tissue reaction			Appearance of palpable lepromata		
Number of days after inoculation	Percent with palpable reactions		Number of weeks after inoculation	Percent with palpable lepromata	
	Inoculated subcutaneously and intravenously	Inoculated subcutaneously only		Inoculated subcutaneously and intravenously	Inoculated subcutaneously only
1.....			1	0	0
2.....	75.9	96.0	2	0	0
3.....			3	0	0
4.....			4	0	10.3
5.....	0	3.5	5	3.4	27.5
6.....			6	10.3	58.6
7.....		0	7	27.5	86.2
8.....			8	68.9	89.6

The results obtained from these studies suggest that the earlier disappearance of the palpable foreign-tissue reaction in the normal well nourished rats and the shorter incubation period in the malnourished rats are due to an interference with the cellular defense mechanism in the malnourished rats. Studies of the numbers of the white blood cells in the circulating blood of malnourished rats have been made to determine whether there has been an interference with the cellular defense mechanism in these rats.

In one experiment the total numbers of white cells in the circulating blood in 5 vitamin B₁ deficient and in 5 control rats were studied. These rats were 1 month old when placed on the experiment. During 15 days on the vitamin B₁ free diet the average number of white blood cells had increased from 5,890 to 8,090, while during the same period the average number in the rats on the control diet had increased from 6,100 to 9,060. It must be remembered that the rats on the vitamin B₁ free diet were but partially depleted during this period as it requires from 2 to 3 weeks to obtain complete depletion. Following subcutaneous inoculation of the deficient rats with an emulsion of a rat leproma there occurred practically no response to the inoculation as shown by the average number of white cells in the circulating blood 1, 2, and 3 weeks after inoculation. The counts were 6,930, 5,120, and 7,300, respectively, while in the control rats there occurred an increase of 50 percent from the time of inoculation to 3 weeks after inoculation.

The counts in the control rats were 9,700, 9,888, and 13,650, respectively.

In another experiment the numbers of white cells in the circulating blood were studied in 5 vitamin B₁ deficient and 5 control rats. These rats were 4 weeks old when placed on the experiment. The results were similar to those of the previous study. During the first 15 days on the vitamin B₁ free diet and before inoculation the average number of white cells had decreased from 8,630 to 7,720, while during the same period the average number in the control rats had increased from 7,400 to 12,210. During a period of 5 weeks following subcutaneous inoculation of an emulsion of a rat leproma the average number of white cells in the deficient rats remained approximately the same, 7,720 and 7,500, while the average number in the control rats had increased 38.5 percent, or from 12,210 to 16,920. From the onset of the experiment to 5 weeks after inoculation, a period of 7 weeks, a decrease of 43.9 percent occurred in the absolute number of lymphocytes in the deficient group and there was an increase of 101.9 percent in the control group of rats.

The results of these two studies suggest that the cellular defense mechanism of the rats is affected by vitamin B₁ deficiency. They particularly suggest that the deficiency affects the lymphocyte forming organs.

Another study showed that this effect on the numbers of white blood cells is not due particularly to the vitamin B₁ deficiency. Identical results were obtained in rats malnourished by means other than vitamin B₁ deficiency. The numbers of white cells in the circulating blood in 10 rats of each of the following groups were determined: (1) Vitamin B₁ deficient rats; (2) poor state of nutrition produced by limiting the intake of the normal control diet; (3) poor state of nutrition produced by limiting the intake of the normal control diet to which had been added 0.4 percent brewer's yeast and 0.2 percent cod-liver oil; and (4) normal control rats.

The average weekly weight of the rats on the three deficient diets was kept between 60 and 80 grams. The control rats gained in weight normally. The average white cell counts were similar in the three deficient groups of rats. During 70 days on the diets the changes that took place in the average numbers of white cells were as follows:

Total number of white blood cells:

Group 1.—Vitamin B₁ deficient, decrease of 30 percent.

Group 2.—Starvation, decrease of 36 percent.

Group 3.—Starvation plus vitamins, decrease of 35 percent.

Group 4.—Control, increase of 0.9 percent.

Absolute number of lymphocytes:

Group 1.—Vitamin B₁ deficient, decreased 37 percent.

Group 2.—Starvation, decreased 38 percent.

Group 3.—Starvation plus vitamins, decreased 34 percent.

Group 4.—Control, increased 5 percent.

Although blood cell counts were made on but few rats in each group, it is believed that they are of significance since the results on repetition were consistent. The studies were made on a total of 35 vitamin B₁ deficient, 30 starvation, and 40 control rats, all males of approximately the same age.

Histological examination made by Senior Pathologist G. L. Fite likewise suggests that vitamin B₁ deficiency interferes with the cellular defense mechanism of the animals. Such examinations were made of tissue from the sites of subcutaneous inoculation of vitamin B₁ deficient and control rats removed on the first, second, fourth, seventh, fourteenth, and twenty-first days after inoculation. It is particularly evident that the control animals displayed a much better response to the foreign tissues and much more rapid healing of the lesions. Thus, on the fourth day the control animals showed beginning fibrosis and organization of the inoculum, whereas the deficient animals showed none, and the lesions of these on the fourth day resembled closely those of the control animals on the second day. On the twenty-first day the control animals showed complete healing of the foreign tissue reaction by organization and fibrosis, while the deficient animals still showed a residue of the inoculum containing large numbers of bacilli. This seems to suggest that the bacilli proliferate well in the detritus of the inoculum in the deficient animals, whereas in the controls this foreign material is more rapidly and readily removed.

The leproma, a granuloma, is believed to be formed by the infiltration of cells and the development of fibrous tissue and is an attempt by the animal organism to overcome the infection. The well nourished animal is better able to attack the infective organism and build up a better defense, hence the larger lesions at the site of subcutaneous inoculation. In the malnourished animal the defense mechanism has been affected and is less able to attack the infecting organism, hence the smaller lesion at the site of subcutaneous inoculation.

From these studies it appears that the shorter incubation period and the smaller lepromata at the site of subcutaneous inoculation in the vitamin B₁ deficient rats were due to an interference with the cellular defense mechanism brought about by a state of general malnutrition and not specifically to vitamin B₁ deficiency.

Gross evidence of generalization of the infection appears earlier in vitamin B₁ deficient than in the normal rats.—The infection was considered generalized when gross lesions were noted in the various organs of the body. Examinations were made either with the unaided eye or with a 6-power hand lens.

In rats receiving subcutaneous inoculations: Thirty-two weeks after subcutaneous inoculation 15 vitamin B₁ deficient and 15 control rats were killed and examined. Twelve, or 80 percent, of the 15 deficient animals had definite gross lesions of the liver and 4, or 26.6 percent, also had definite gross lesions of the spleen. None of the 15 control rats had lesions other than at the site of the subcutaneous inoculation. At this time no gross lesions were noted in any organs other than the liver and spleen.

Twenty days after the first examination, 8 months and 1 day after inoculation, 16 rats of each group were killed and examined. Eleven, or 68.7 percent, of the 16 deficient rats had gross evidence of a generalized infection, 4 had gross lesions of the skin, 11 of the liver, 6 of the spleen, 1 of the pericardium, 1 of the bony skeleton, and 2 had a generalized adenopathy. None of the 16 normal control rats had definite gross evidence of a generalized infection.

A comparison of the findings noted at the second examination with those noted at the first, 20 days intervening, is interesting. At the first examination the only lesions noted, signifying a generalized infection, were those of the liver and spleen, while at the second examination lesions were noted in the skin, pericardium, and bony skeleton.

Combining the findings observed at both examinations, gross evidence of a generalized infection was noted in 23, or 74.1 percent, of the 31 deficient rats, while none of the 31 well nourished control rats had such evidence.

A repetition of this experiment produced similar results. Nine months after subcutaneous inoculation 12 vitamin B₁ deficient and 12 normal rats were examined. Four, or 33.3 percent, of the deficient rats had definite gross lesions of the liver, and 2 of these also had gross lesions of the spleen. None of the 12 well nourished control rats had such lesions.

It will be noted that there were fewer rats in the second than in the first experiment with gross evidence of a generalized infection, although they were examined at approximately the same time after inoculation. It is not correct to compare the results in two experiments such as these, since it is impossible to determine the exact amount of infective material inoculated, which might be entirely different in the two experiments. However, in this instance the difference might be explained by the fact that the Hawaiian¹ strain of

¹ Isolated from a wild rat in Honolulu, Hawaii.

rat leprosy was employed in the first experiment and the Florida² strain in the second. The Hawaiian strain is apparently more virulent than the Florida strain.³

In rats receiving intraperitoneal inoculations: Following intraperitoneal inoculation the first lesions noted are those of the omentum.

Although the organisms early gain entrance to the lymph and blood streams and become disseminated throughout the animal, gross lesions in organs other than the omentum and mesentery are noted earlier in the vitamin B₁ deficient than in the control animals.

In the first experiment in which the rats were inoculated intraperitoneally, animals were examined at 8, 15, and 20 weeks after inoculation in order to determine at what time gross lesions developed in the various organs. Twelve rats from each group were examined 8 weeks after inoculation and 5 at 15 weeks. Lesions were noted only in the omentum and mesentery. Twenty weeks after inoculation 6 deficient and 7 control rats were living. On examination gross lesions were seen in the liver and spleen of each, and of the skin in 4 of the deficient animals, and in none of the control animals.

In a repetition of this experiment, 13 vitamin B₁ deficient and 13 control rats were examined 20 weeks after inoculation. Of the deficient rats, 46.1 percent had gross lesions of the skin, 84.6 percent of the spleen, and 100 percent of the liver. Of the normal rats, 53.8 percent had gross lesions of the spleen. None had gross lesions of the skin or liver. The lesions of the spleens of the deficient rats were more numerous and larger than those in the control rats.

Intraperitoneal inoculations were again repeated. Twenty weeks after inoculation 5 rats of each group were examined. Of the 5 vitamin B₁ deficient rats, 4 had gross lesions of the liver and 1 of the spleen, while none of the 5 controls had such lesions. Twenty-four weeks after inoculation, 3 of 4 deficient and none of 4 control animals had gross lesions of the abdominal organs. Six months after inoculation the rats remaining alive were examined. Seven of 9 deficient and none of 17 control rats had gross lesions of the liver or liver and spleen.

At the three examinations, 14, or 77.7 percent, of the total of 18 deficient rats had gross lesions of the liver or liver and spleen, while none of the 26 control rats had such lesions. At each examination the lesions of the omentum and mesentery were considerably larger in the deficient than in the normal control animals.

In the first experiment in which lesions were noted in the skin in addition to those in the liver and spleen, 20 weeks after inoculation, the Hawaiian strain of rat leprosy was employed, while in the third, in which no such lesions occurred, the Florida strain was employed.

² Isolated from a wild rat in Jacksonville, Fla.

³ Bulletin 173, National Institute of Health, U. S. Public Health Service, 1940.

In rats receiving intravenous inoculations: Following intravenous inoculation the organisms are immediately spread throughout the animal tissues and the infection is generalized from the onset. However, definite gross lesions develop earlier in the vitamin B₁ deficient than in the normal rats.

Twenty-three vitamin B₁ deficient and 22 control rats were examined 16 weeks after intravenous inoculation. Of the 23 deficient rats, 22, or 95.6 percent, had gross lesions of the liver, while none of the 22 controls had such lesions. Lesions of the liver were the only ones noted at this time.

Intravenous inoculations were repeated. Sixteen weeks after inoculation, 20 vitamin B₁ deficient and 20 control rats were examined. Of the 20 deficient rats, 20, or 100 percent, had gross lesions of the liver and 2, or 10 percent, of the spleen. Of the 20 control rats, 8, or 40 percent, had gross lesions of the spleen or spleen and liver.

Six months after inoculation 14 of the control rats were living. Of these 14 rats, 8, or 57.1 percent, had gross lesions of the liver, 13, or 92.8 percent, of the spleen, and 14, or 100 percent, of either the liver or spleen or both organs. Thus, at 16 weeks 100 percent of the deficient rats had gross lesions of the liver or liver and spleen, while but 40 percent of the control rats had such lesions. At 6 months 100 percent of the control rats had such lesions.

The results of these two experiments and those of several subsequent experiments, in which the inoculations were made by the intravenous route, are tabulated in table 5.

In rats receiving intranasal instillations: Four weeks following intranasal instillation smears prepared by crushing cervical lymph glands of 16 vitamin B₁ deficient and 9 control rats, when examined, revealed no acid-fast organisms.

Smears similarly prepared with cervical glands of 21 vitamin B₁ deficient and 15 control rats were examined 8 weeks after instillation. Acid-fast organisms were observed in 19, or 90.4 percent, of the smears prepared from the glands of the deficient rats and in 9, or 60 percent, of those prepared from the normal or control rats.

Approximately 10 weeks after instillation acid-fast organisms were demonstrated in smears prepared with cervical lymph glands of each of 8 vitamin B₁ deficient rats and in but 50 percent of the smears similarly prepared from 8 control rats.

Nine months after instillation 8 of the vitamin B₁ deficient and 6 normal control rats were examined for gross evidences of generalization of the infection. Of the 8 deficient rats, 6, or 75 percent, had extension into the periglandular tissues of the cervical region; 2, or 25 percent, had generalized adenopathy; 6, or 75 percent, had gross lesions of the liver; 4, or 40 percent, of the spleen; and 1, or 12.5

percent, of the skin. In none of the 8 control rats was there noted any gross evidence of generalization of the infection.

From these experiments it can be definitely stated that gross evidence of a generalized infection appears earlier in the vitamin B₁ deficient than in the normal control rats.

In the course of these experiments it was thought advisable to study the relation of calcium deficiency to rat leprosy.⁴

TABLE 5.—Gross evidence of a generalized infection in vitamin B₁ deficient and normal rats

Mode of inoculation	Experiment No.	Number of weeks after inoculation	Group of rats	Number of rats	Number and percent of rats showing gross lesions of the liver, spleen, or skin			Total rats with gross evidence of generalized infection
					Liver	Spleen	Skin	
Subcutaneous	XVIII	32 to 35	Vitamin B ₁ deficient	31	23 or 74.1 percent.	10 or 32.2 percent.	4 or 12.9 percent.	23 or 74.1 percent.
			Control	31	None	None	None	None
	XXVIII	39	Vitamin B ₁ deficient	12	4 or 33.3 percent.	2 or 16.6 percent.	None	4 or 33.3 percent.
			Control	12	None	None	None	None
Intraperitoneal	V	20	Vitamin B ₁ deficient	6	6 or 100.0 percent.	6 or 100.0 percent.	4 or 66.6 percent.	6 or 100.0 percent.
			Control	7	None	None	None	None
	XV	20	Vitamin B ₁ deficient	13	13 or 100.0 percent.	11 or 84.6 percent.	6 or 46.1 percent.	13 or 100.0 percent.
			Control	13	None	7 or 53.8 percent.	None	7 or 53.8 percent.
	XXX	27	Vitamin B ₁ deficient	9	7 or 77.7 percent.	4 or 44.4 percent.	None	8 or 88.3 percent.
			Control	17	None	None	None	None
Intravenous	XXI	15	Vitamin B ₁ deficient	23	22 or 95.6 percent.	None	None	22 or 95.6 percent.
			Control	22	None	None	None	None
	XXXI	16	Vitamin B ₁ deficient	20	20 or 100.0 percent.	2 or 10 percent.	None	20 or 100.0 percent.
			Control	20	6 or 30.0 percent.	2 or 10 percent.	None	8 or 40.0 percent.
	XL	16 to 17	Vitamin B ₁ deficient	22	20 or 90.9 percent.	3 or 13.6 percent.	None	20 or 90.9 percent.
			Control	22	None	None	None	None
Intranasal	XII	40	Vitamin B ₁ deficient	8	6 or 75.0 percent.	6 or 75.0 percent.	2 or 25 percent.	6 or 75.0 percent.
			Control	8	None	None	None	None

In the first experiment 3 groups of 34 rats each were studied: Vitamin B₁ deficient, calcium deficient, and normal control. Examinations were made 17 weeks after inoculation. Of 22 rats of the vitamin B₁ deficient and calcium deficient groups, 90.9 percent had gross evidence of generalization of the infection and none of 22 controls had evidence of such infection.

In another experiment calcium deficient rats were again studied. Examinations were made at approximately 17 weeks after inoculation, at which time 100 percent of 16 calcium deficient rats and 6.2 percent of 26 control rats had gross evidence of generalization of the infection.

⁴ In the remaining experiments intravenous inoculations only have been employed.

The results of these two experiments suggest that calcium deficient rats are as susceptible to rat leprosy as are vitamin B₁ deficient rats. The questions arose: Is the increased susceptibility of the vitamin B₁ deficient rats due specifically to a deficiency in that vitamin, and that of the calcium deficient specifically to a deficiency in that element or is there a factor common to both which produces the increased susceptibility?

To answer these questions experiments were conducted, the results of which have been reported (6). The rats maintained on the vitamin B₁ deficient diet were not found to be deficient in calcium, as shown by chemical analysis of the tails. The rats maintained on the calcium deficient diet were found to be deficient in vitamin B₁, as shown by the determinations of the amount of that vitamin in the blood. The administration of vitamin B₁ to both groups, those maintained on the vitamin B₁ deficient and those maintained on the calcium deficient diet, caused them to be no more susceptible to the infection than the normal control rats. These findings strongly suggest that the increased susceptibility is due to vitamin B₁ deficiency and not to calcium deficiency and that, for some reason as yet undetermined, the rats maintained on the calcium deficient diet are incapable of utilizing the vitamin B₁ available in the diet. Evidence has been obtained which suggests that the calcium deficient diet employed is also deficient in some other factor or factors.

SUMMARY AND CONCLUSIONS

The incubation period of rat leprosy in rats maintained on a vitamin B₁ free diet is definitely shorter than in rats maintained on a normal control diet. Evidence has been obtained which suggests that the shortened incubation period is due to an interference with the cellular defense mechanism of the animal rather than specifically to the vitamin deficiency.

Rats maintained on a vitamin B₁ deficient diet are definitely more susceptible to rat leprosy than are normal rats. The criteria for susceptibility employed have been the development of gross evidence of generalization of the infection. Evidence has been obtained which suggests that the increased susceptibility is due specifically to the vitamin B₁ deficiency. Rats maintained on the deficient diet which received purified vitamin B₁ as a supplement are no more susceptible than are normal rats.

Rats maintained on a calcium deficient diet are approximately as susceptible as those maintained on the vitamin B₁ free diet. Evidence has been obtained which has shown the rats maintained on the calcium deficient diet to be deficient also in vitamin B₁ through the inability to utilize the vitamin B₁ available in the diet. The amount of vitamin B₁ in the circulating blood of the rats maintained on the calcium

deficient diet is approximately the same as that of the rats maintained on the vitamin B₁ free diet. Rats maintained on the calcium deficient diet which receive purified B₁ as a supplement are no more susceptible than are normal rats.

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CHRONIC MANGANESE POISONING

A Review

In 1937 several patients with an obscure, disabling, neurologic disease were studied in the University of Pennsylvania Hospital and were found to have chronic manganese poisoning. The processors of manganese ore at the mill at which these men had been employed as laborers requested the Public Health Service to study the health hazards in that industry in the hope that the cause of several cases of manganese poisoning among the employees might be ascertained, and that preventive measures might be instituted to obviate the further occurrence of this disease. Accordingly, in August 1937 the Public Health Service in cooperation with the Division of Industrial Hygiene of the Pennsylvania Department of Health began medical, engineering, and chemical studies in two manganese-ore-crushing mills to determine, among other things, the concentration of manganese dusts in the air at the breathing level in all parts of the plant where men work. The report of these investigations has been published recently as Public Health Bulletin No. 247.¹ One of these mills had been in operation about 20 years and in this mill little attempt had been made to prevent the dispersal of dust. The highest manganese con-

¹ Public Health Bulletin No. 247, Chronic Manganese Poisoning in an Ore-Crushing Mill, by Robert H. Flinn, Paul A. Neal, Warren H. Reinhart, J. M. DallaValle, William B. Fulton, and Allan E. Dooley. This report contains a chapter by J. W. Miller, reporting post-mortem findings in a case of manganese poisoning, and a chapter by Lawrence T. Fairhall, concerning chemical analysis of the manganese content of the blood. The report contains a description of the sources and uses of manganese, manufacturing methods, results of engineering and medical studies, and detailed case histories of 6 of the 11 reported cases of manganese poisoning. There is an annotated bibliography of 42 titles and an analytical index. The Bulletin contains 77 pages. It is available from the Superintendent of Documents, Government Printing Office, Washington, D. C., at 15¢ per copy.

centrations were found in the vicinity of the pulverizer, where the average manganese concentration amounted to 173 mg. of manganese per cubic meter. In a newly completed plant, the machinery was enclosed, ore was transported from place to place by a crane or on conveyor belts, and exhaust ventilation was applied at strategic points. The dustiest operation in the latter plant, filling bags or barrels, exposed the operators to 6 mg. per cubic meter, and the average exposure throughout the plant was little more than 2 mg. per cubic meter.

Chronic manganese poisoning was the principal disease found on medical examination of 34 men employed, or formerly employed, in the older of these two plants. Eleven cases of this disease were found. The prevalence of the disease depended on the amount of manganese in the workroom air and on the duration of employment. No cases were found among men exposed to less than 30 mg. of manganese per cubic meter, but this should not be considered as a threshold limit because only a few men were so exposed, and some exposures were intermittent. Five of the 6 men exposed for more than 3 years to concentrations in excess of 90 mg. per cubic meter had chronic manganese poisoning. In one man, manganese poisoning developed after 10 months' exposure to 30 mg. per cubic meter, and in another case, after a year's exposure to 50 mg. of manganese per cubic meter.

The disease is characterized by muscular stiffness and incoordination which progresses until disability results. It is usually first apparent as disturbances in gait and difficulty in stepping backward without falling down, speech disturbances, including stuttering and running together of words, muscular twitchings or tremors, and occasionally a masklike facial expression. The worker often complains of extreme drowsiness, weakness, or lassitude, muscular twitchings and cramps, and difficulty in walking and talking.

Chemical analysis of viscera obtained at post-mortem examination from one of these cases of manganese poisoning showed 3.07 and 4.87 mg. of manganese per 100 g. of dry tissue in the right and left lung, respectively, and very much smaller amounts in the other organs, indicating that the patient still had a reservoir of manganese in the lungs 7 years after his last exposure to this dust. Also, a few months before death he had been found to be excreting small amounts of manganese in the urine.

Extensive laboratory examinations of these workers indicated that a low white-cell count with a decreased percentage of neutrophils, a slightly lowered blood calcium content, and a slight reduction in the middle zone of Lange's test were often associated with manganese poisoning. A lowered hemoglobin content was observed in several of the manganese workers. Analysis of the blood for manganese content was of no assistance in this study. Urinary manganese appears to reflect a present or past exposure to manganese compounds rather

than evidence of intoxication. Impairment of kidney or liver function was not observed.

The differential diagnosis of this disease, which has many features in common with multiple sclerosis, paralysis agitans, and progressive lenticular degeneration, has been discussed in this bulletin.

The control methods which have come into general use in other dusty trades, namely, enclosed processes, mechanical conveyors, and exhaust ventilation, have been tested and were found to be effective in preventing the dispersal of manganese dusts. To supplement this medical examinations, made quarterly, should make it possible to detect early cases of manganese poisoning, to permit their transfer to nondusty occupations, and to direct attention to faulty control methods. Especial attention should be given to complaints of general weakness, drowsiness, and muscular twitchings and cramps. Disorders in gait and speech or the occurrence of tremor suggest the onset of this disease. White blood cell counts may well be made, as it seems that a reduction in these values is associated with manganese poisoning, although it is not known whether these findings precede the development of physical findings. Inasmuch as disability resulting from well-advanced stages of manganese poisoning is permanent, every effort should be made to detect and transfer workers away from a hazardous exposure at the onset of the disease until the manganese hazard has been controlled.

PSITTACOSIS CASE IN IDAHO TRACED TO SHELL PARAKEETS SHIPPED FROM SOUTHERN CALIFORNIA AVIARY

According to information received under date of May 14, from Dr. W. M. Dickie, Director of Public Health of California, the source of infection in the fatal human case of psittacosis which occurred in Caldwell, Idaho, in December 1939, was shell parakeets from a pet shop in Los Angeles, Calif.

Investigation has revealed that a pair of shell parakeets had been purchased by the deceased in the latter part of November from a local dealer in Caldwell, Idaho, whose only stock of parakeets was a shipment of 24 birds received earlier in the month from a pet shop in Los Angeles. The purchaser became ill on December 5 and died on December 18. Psittacosis infection was subsequently proved in the birds purchased by the deceased and in the remaining birds held by the Caldwell dealer. The infection was also proved in several shell parakeets and other psittacine birds in the Los Angeles pet shop.

As no infection was found in the local breeding aviaries which supplied the California dealer, and as this dealer had imported from other countries larger psittacine birds, several shipments of which had been found to be infected, it is probable that the infection was introduced into the Los Angeles pet shop by imported birds.

Under the direction and supervision of representatives of the California State Department of Health and the Los Angeles County Health Department, all psittacine birds of the Los Angeles pet shop were destroyed and the premises were cleaned, disinfected, and remodeled.

Birds from the same Los Angeles pet shop were apparently involved in an outbreak of 3 cases of psittacosis in Tucson, Ariz., in October and November 1939.¹ Two cases occurred in young adults of the same family and 1 case in a nurse who cared for the two patients.

The family owned 2 lovebirds, purchased from a local dealer in July 1938,² which had been procured from the Los Angeles pet shop in question in April 1938.² When psittacosis was suggested, the birds were set free and were therefore not available for examination; but examination of the other birds of the same group in possession of the local dealer is stated to have been negative. During 7 weeks in the summer of 1939 the two lovebirds had been cared for by a family which owned 6 canaries; it was stated that these canaries had shown no evidence of illness and that the canaries and lovebirds were kept in separate rooms. The lovebirds were returned to the owner about September 15, 1939. The source of the infection in the two lovebirds was not determined.

In this connection, it is of interest to note that, in an outbreak of psittacosis in Pasadena, Calif., some years ago, it was reported that one case was traced to canaries which had shown no evidence of illness, but when killed and examined were found to be infected.

COURT DECISION ON PUBLIC HEALTH

City ordinance pertaining to public market places upheld.—(Florida Supreme Court; *McCroan v. Bloodworth, Chief of Police, et al.*, 193 So. 431; decided January 23, 1940.) An ordinance of the city of Apalachicola provided, in section 1, for the designation, when deemed practicable, of parts of city streets as public market places for the sale of meat, fruit, vegetables, or other foodstuffs and for the allotting to each permittee of one uniform space or more as required, each space to be sufficient to accommodate one truck or automobile. The remainder of the ordinance dealt with the securing and revocation of, and fees for, permits; the inspection of foodstuffs; the prohibition of the sale of unwholesome foodstuffs; and the prohibition, except as otherwise expressly provided, of the sale of foodstuffs at other than the allotted spaces.

¹ Public Health Reports, Jan. 12, 1940, p. 99.

² First report (Dec. 27, 1939) from Dr. J. D. Dunshee, of the Arizona State Board of Health, stated the birds had been purchased by the local dealer in April 1939, while a later report (Jan. 9, 1940) gives the dates of purchase and resale as here recorded.

In habeas corpus proceedings the petitioner challenged the provisions of the ordinance with the exception of section 1, but the supreme court said that it appeared that the provisions complained of were authorized by the city charter act and did not violate any provisions of the State constitution or statutes. The court further stated that the provisions came within the purview of the State police power and were properly declared to be for the protection of the health and welfare of the people of the city.

DEATHS DURING WEEK ENDED MAY 25, 1940

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended May 25, 1940	Correspond- ing week, 1939
Data from 88 large cities of the United States:		
Total deaths.....	8,280	8,019
Average for 3 prior years.....	8,232	
Total deaths, first 21 weeks of year.....	191,991	191,396
Deaths under 1 year of age.....	490	491
Average for 3 prior years.....	504	
Deaths under 1 year of age, first 21 weeks of year.....	10,705	11,206
Data from industrial insurance companies:		
Policies in force.....	65,481,168	67,244,634
Number of death claims.....	12,309	12,689
Death claims per 1,000 policies in force, annual rate.....	9.8	9.8
Death claims per 1,000 policies, first 21 weeks of year, annual rate.....	10.5	11.6

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED JUNE 1, 1940

Summary

The incidence of each of the 9 communicable diseases included in the weekly telegraphic reports published in the following table, with the exception of poliomyelitis and typhoid fever, showed a decrease as compared with the preceding week and, with the exception of influenza and poliomyelitis, was below the 5-year (1935-39) median expectancy. The accumulative total to date for each of these diseases, except influenza and poliomyelitis, is also below the 5-year cumulative median.

Of the 47 cases of poliomyelitis reported for the current week, 35 cases occurred in the 3 Pacific States—Washington 25 cases (all in Pierce county, 13 in Tacoma), California 9 (5 in Los Angeles), and 1 case in Oregon. During the preceding week Washington reported 10 cases, and the week before that 7 cases. Since the first of the year, a total of 544 cases of poliomyelitis has been reported in the United States, of which 163 cases occurred in the 3 Pacific States (112 cases in California).

During the current week, 147 cases of typhoid fever were reported (20 cases in Tennessee, 11 in Georgia, and 10 in Missouri), as compared with 141 cases for the preceding week. The current incidence for the country as a whole, however, is below the 5-year median expectancy of 197 cases.

Of 18 cases of Rocky Mountain spotted fever reported currently, 2 cases occurred in Maryland and 1 case in Virginia. The other cases were reported from the northwestern States.

Sixteen cases of endemic typhus fever were reported from the South Atlantic and South Central States.

For the current week the Bureau of the Census reports 7,682 deaths in 88 major cities of the United States, as compared with 8,280 for the preceding week and with a 3-year (1937-39) average of 8,232 for the corresponding week.

Telegraphic morbidity reports from State health officers for the week ended June 1, 1940, and comparison with corresponding week of 1939 and 5-year median

In these tables a zero indicates a definite report, while leaders imply that, although none were reported, cases may have occurred.

Division and State	Diphtheria			Influenza			Measles			Meningitis, meningococcus		
	Week ended		Median, 1935-39	Week ended		Median, 1935-39	Week ended		Median, 1935-39	Week ended		Median, 1935-39
	June 1, 1940	June 3, 1939		June 1, 1940	June 3, 1939		June 1, 1940	June 3, 1939		June 1, 1940	June 3, 1939	
NEW ENGL.												
Maine	0	0	0				344	70	105	0	0	0
New Hampshire	0	0	0				11	5	8	0	0	0
Vermont	0	0	0				2	130	79	0	0	0
Massachusetts	3	2	3				1,185	976	647	1	2	3
Rhode Island	0	0	0				193	136	81	0	0	0
Connecticut	0	0	2	1	1	1	26	579	203	0	0	0
MID. ATL.												
New York	15	20	23	16	16	16	919	2,150	2,430	1	5	6
New Jersey	4	12	9	4		5	990	36	724	0	2	2
Pennsylvania	14	17	24				454	57	1,846	2	18	9
E. NO. CEN.												
Ohio	8	15	21	38	13	22	43	96	1,491	1	3	5
Indiana	5	4	7	4	1	9	22	10	215	0	0	0
Illinois	13	23	32	18	8	15	163	35	454	1	4	4
Michigan ¹	1	8	7	11	4	2	610	403	403	2	0	2
Wisconsin	2	2	2	19	35	27	1,005	743	743	0	0	0
W. NO. CEN.												
Minnesota	1	2	4	1	2	1	46	216	279	0	0	0
Iowa	1	3	2		2	2	145	188	188	0	1	0
Missouri	2	3	11	1		36	53	4	71	0	0	2
North Dakota	1	0	0		47	6	5	14	14	1	0	0
South Dakota	0	0	1		8		0	196	4	0	0	0
Nebraska	6	0	2		6	1	17	160	160	1	0	0
Kansas	1	0	3	1	5		407	58	58	0	0	0
SO. ATL.												
Delaware	0	0	0				1	36	17	0	0	0
Maryland ^{2,3}	1	2	3	1	6	2	4	129	129	0	1	1
Dist. of Col.	3	4	6			1	2	334	110	0	0	2
Virginia ³	3	15	10	48	87		227	999	390	1	0	2
West Virginia ¹	3	7	7	5	4	11	15	7	59	1	1	3
North Carolina	8	7	7	6	1	1	157	459	309	0	0	3
South Carolina ⁴	9	3	2	128	188	77	20	15	64	1	3	1
Georgia ⁴	3	7	3	14	51		73	106	0	1	0	0
Florida ⁴	6	3	4		18	3	116	73	20	0	0	1
E. SO. CEN.												
Kentucky	2	4	4	49	2	3	79	11	105	0	0	5
Tennessee	3	3	3	19	20	20	153	82	82	0	1	3
Alabama ⁴	4	3	10	14	32	27	89	148	103	3	3	3
Mississippi ²	3	8	5						0	0	0	0
W. SO. CEN.												
Arkansas	3	2	3	6	48	28	108	28	28	0	1	0
Louisiana ⁴	5	10	8	19	4	4	3	79	32	1	0	0
Oklahoma ⁴	3	2	8	10	22	25	15	160	49	1	0	0
Texas ⁴	15	10	32	121	86	100	1,058	420	280	0	1	3
MOUNTAIN												
Montana ³	0	0	0	6	3		31	124	90	0	1	1
Idaho ³	0	1	0				37	55	16	0	0	0
Wyoming ³	0	1	0				24	26	16	1	0	0
Colorado ^{3,5}	9	6	6	8	7		43	151	151	0	0	0
New Mexico	1	0	1		1		56	14	16	0	0	0
Arizona	2	0	1	40	27	21	35	13	33	0	0	0
Utah ^{2,5}	0	2	0		1		479	86	80	0	0	0
PACIFIC												
Washington	1	5	1				320	777	339	0	0	0
Oregon ³	4	0	0	5	24	15	345	74	74	0	0	1
California	15	20	21	19	34	34	354	2,085	1,281	0	2	2
Total	183	236	339	622	804	570	10,484	12,783	14,169	20	49	96
22 weeks	7,032	9,267	11,032	164,674	147,113	136,490	172,434	301,185	301,185	868	1,052	3,134

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended June 1, 1940, and comparison with corresponding week of 1939 and 5-year median—Con.

Division and State	Poliomyelitis			Scarlet fever			Smallpox			Typhoid and paratyphoid fever		
	Week ended		Median, 1935-39	Week ended		Median, 1935-39	Week ended		Median, 1935-39	Week ended		Median, 1935-39
	June 1, 1940	June 3, 1939		June 1, 1940	June 3, 1939		June 1, 1940	June 3, 1939		June 1, 1940	June 3, 1939	
NEW ENG.												
Maine.....	0	0	0	10	4	10	0	0	0	0	0	1
New Hampshire.....	0	0	0	1	5	20	0	0	0	0	0	0
Vermont.....	0	0	0	2	12	6	0	0	0	0	0	0
Massachusetts.....	0	0	0	101	138	217	0	0	0	4	0	1
Rhode Island.....	0	0	0	4	8	23	0	0	0	0	0	0
Connecticut.....	0	1	0	52	35	73	0	0	0	4	1	2
MID. ATL.												
New York.....	0	2	1	661	362	566	0	20	0	4	6	6
New Jersey.....	0	1	0	284	119	131	0	0	0	2	3	3
Pennsylvania.....	1	0	0	271	194	338	0	0	0	9	7	7
E. NO. CEN.												
Ohio.....	2	0	0	369	267	267	0	28	2	9	16	7
Indiana.....	0	0	0	87	78	88	1	22	22	5	1	1
Illinois.....	0	2	0	650	277	401	9	10	15	2	7	6
Michigan ¹	0	0	0	238	262	271	0	7	1	0	1	5
Wisconsin.....	0	1	0	109	103	207	3	1	2	1	1	1
W. NO. CEN.												
Minnesota.....	0	0	0	55	70	117	7	17	16	0	1	1
Iowa.....	1	0	0	22	45	68	3	30	22	0	2	0
Missouri.....	0	0	0	38	41	91	0	28	28	10	1	8
North Dakota.....	0	0	0	2	2	23	0	0	11	4	4	2
South Dakota.....	0	0	0	2	15	15	1	14	14	2	0	0
Nebraska.....	0	0	0	6	4	38	1	9	9	0	0	0
Kansas.....	0	0	0	42	38	57	1	6	13	2	0	1
SO. ATL.												
Delaware.....	0	0	0	2	5	3	0	0	0	0	0	0
Maryland ²	0	0	0	33	15	38	0	0	0	1	2	4
Dist. of Col.....	0	0	0	20	7	11	0	0	0	1	1	1
Virginia ³	0	0	0	36	19	20	0	0	0	0	6	7
West Virginia ¹	0	0	1	20	26	40	0	0	0	3	6	5
South Carolina.....	0	1	2	20	13	14	0	1	4	4	9	7
Georgia ⁴	1	22	1	1	5	5	0	0	0	3	9	9
Florida ⁴	0	1	1	10	6	6	0	12	0	11	17	7
Florida ⁴	0	1	1	5	9	4	1	0	0	3	2	2
E. SO. CEN.												
Kentucky.....	0	0	0	37	19	19	1	1	0	5	5	5
Tennessee.....	0	0	0	44	38	18	3	55	1	20	9	9
Alabama ⁴	0	1	1	7	10	6	4	0	0	2	10	7
Mississippi ²	1	0	0	5	1	2	0	0	0	1	3	3
W. SO. CEN.												
Arkansas.....	0	0	0	11	4	4	4	5	2	4	11	10
Louisiana ⁴	1	0	1	7	2	6	1	0	0	7	10	10
Oklahoma ⁴	0	0	0	10	10	19	1	40	8	5	7	7
Texas ⁴	2	2	0	19	30	50	3	7	13	8	12	12
MOUNTAIN³												
Montana.....	0	0	0	5	14	12	0	2	5	1	0	1
Idaho ³	3	0	0	4	2	6	0	0	1	1	0	0
Wyoming ³	0	0	0	3	4	8	1	0	3	0	0	0
Colorado ^{3,4}	0	0	0	22	33	37	1	6	3	2	1	1
New Mexico.....	0	0	0	2	4	9	0	0	0	0	0	1
Arizona.....	0	7	0	0	4	13	0	3	0	0	1	2
Utah ^{3,4}	0	0	0	9	23	23	0	1	0	0	3	0
PACIFIC												
Washington.....	25	0	0	40	29	29	0	3	3	2	28	1
Oregon ³	1	1	0	6	11	23	0	7	7	0	0	3
California.....	9	17	5	92	137	175	1	2	8	5	7	7
Total.....	47	60	36	3,476	2,559	4,379	47	337	242	147	210	197
22 weeks.....	544	511	468	104,165	103,808	145,153	1,545	7,618	6,750	1,958	2,720	2,720

See footnotes at end of table.

Telegraphic morbidity reports from State health officers for the week ended June 1, 1940, and comparison with corresponding week of 1939 and 5-year median—Con.

Division and State	Whooping cough		Division and State	Whooping cough	
	Week ended—			Week ended—	
	June 1, 1940	June 3, 1939		June 1, 1940	June 3, 1939
NEW ENG.			SO. ATL.—continued		
Maine.....	12	27	South Carolina ⁴	24	69
New Hampshire.....	16	0	Georgia ⁴	17	48
Vermont.....	7	39	Florida ⁴	2	47
Massachusetts.....	130	109	E. SO. CEN.		
Rhode Island.....	0	43	Kentucky.....	72	15
Connecticut.....	33	45	Tennessee.....	33	70
MID. ATL.			Alabama.....	11	103
New York.....	343	334	Mississippi ¹		
New Jersey.....	58	294	W. SO. CEN.		
Pennsylvania.....	238	194	Arkansas.....	15	20
E. NO. CEN.			Louisiana ⁴	48	6
Ohio.....	269	169	Oklahoma ⁴	10	3
Indiana.....	16	71	Texas ⁴	391	129
Illinois.....	50	233	MOUNTAIN		
Michigan ²	217	163	Montana ³	4	6
Wisconsin.....	60	140	Idaho ³	12	7
W. NO. CEN.			Wyoming ³	1	0
Minnesota.....	41	43	Colorado ³ ⁵	20	28
Iowa.....	25	25	New Mexico.....	14	18
Missouri.....	28	22	Arizona.....	42	5
North Dakota.....	5	1	Utah ³ ⁵	147	47
South Dakota.....	3	1	PACIFIC		
Nebraska.....	12	7	Washington.....	41	16
Kansas.....	30	28	Oregon ³	36	17
SO. ATL.			California.....	385	181
Delaware.....	6	20	Total.....	3,292	3,268
Maryland ² ³	81	35	22 weeks.....	69,784	87,076
Dist. of Col.....	8	27			
Virginia ³	68	128			
West Virginia ¹	83	33			
North Carolina.....	107	197			

¹ New York City only.

² Period ended earlier than Saturday.

³ Rocky Mountain spotted fever, week ended June 1, 1940, 18 cases as follows: Maryland, 2; Virginia, 1; Montana, 3; Idaho, 2; Wyoming, 4; Colorado, 2; Utah, 3; Oregon, 1.

⁴ Typhus fever, week ended June 1, 1940, 16 cases as follows: South Carolina, 3; Georgia, 5; Florida, 4; Alabama, 2; Louisiana, 1; Oklahoma, 1; Texas, 1.

⁵ Colorado tick fever, week ended June 1, 1940, Colorado, 9 cases.

VENEREAL DISEASES
New Cases Reported for March 1940¹
Reports from States

	Syphilis								Gonorrhea		Other venereal diseases		
	Early			Late		Congenital		All Syphilis ¹		Number	Rate per 10,000 population	Number	Rate per 10,000 population
	Primary and secondary	Early latent *	Rate per 10,000 population	Includes late-latent	Rate per 10,000 population	Number	Rate per 10,000 population	Number	Rate per 10,000 population				
Alabama	242	180	1.44	201	0.69	49	0.17	1,366	4.67	319	1.09	4	0.01
Alaska ⁴													
Arizona	27	22	1.17	45	1.68	26	.62	230	5.50	142	3.40	1	.02
Arkansas	215	210	2.05	598	2.88	24	.12	1,160	5.59	147	.71	6	.03
California ⁴													
Colorado	82		.76	151	1.40	17	.16	250	2.32	109	1.01		
Connecticut	16	5	.12	89	.51	12	.07	162	.93	101	.58		
Delaware	11	19	1.14	23	.87	5	.19	140	5.32	23	.87		
District of Columbia								631	9.92	272	4.28	6	.09
Florida	8	470	2.81	1,104	6.50	61	.36	1,787	10.52	136	.80	8	.05
Georgia		1,232	3.96	864	2.78			2,096	6.73	74	.24	7	.02
Hawaii	4	3	.17	27	.67	6	.15	56	1.38	51	1.26		
Idaho	11		.22	25	.50	2	.04	41	.82	15	.30		
Illinois	119	422	.68	1,340	1.69	86	.11	1,967	2.48	1,247	1.58	42	.05
Indiana	95	31	.36	278	.80	18	.05	570	1.63	138	.40	3	.01
Iowa	57	54	.43	123	.48	21	.08	267	1.04	126	.49		
Kansas	45	32	.41	78	.42	18	.10	211	1.13	73	.39		
Kentucky	98	16	.39	278	.94	17	.06	590	1.99	312	1.05	3	.01
Louisiana	206		.96					511	2.38	101	.47	3	.01
Maine	18		.21	13	.15	5	.06	36	.42	29	.34		
Maryland	92	23	.68	249	1.48	11	.07	950	5.64	305	1.81	17	.10
Massachusetts	64		.14	419	.95	18	.04	501	1.13	308	.70		
Michigan	83	98	.37	433	.89	44	.09	813	1.67	573	1.17	18	.04
Minnesota	22	20	.16	163	.63	5	.02	215	.80	171	.64		
Mississippi	503	967	7.30	806	3.95	83	.41	5,276	25.86	2,271	11.13		
Missouri	162	375	1.24	250	.62	41	.10	856	2.13	203	.50	5	.01
Montana	12		.22	23	.42	2	.04	42	.77	20	.37		
Nebraska	27	16	.32	25	.18	1	.01	69	.51	50	.37		
Nevada	1	4	.39	8	.78	3	.29	15	1.47	16	1.57		
New Hampshire		2	.06	5	.10	2	.04	19	.37	5	.10		
New Jersey	121	129	.80	581	1.33	43	.10	1,013	2.32	230	.53	1	.002
New Mexico	28		.66	78	1.85	9	.21	115	2.79	59	1.40		
New York	353	320	.62	2,773	2.13	176	.14	3,874	2.98	1,547	1.19	18	.01
North Carolina	283	964	3.53	862	2.44	65	.18	2,174	6.16	302	.86	21	.06
North Dakota ⁴													
Ohio ⁴								833	3.24	254	.99		
Oklahoma								92	.89	83	.80		
Oregon	21	14	.34	47	.45	5	.05	92	.89	83	.80		
Pennsylvania	345	969	1.29	255	.25	82	.08	1,651	1.61				
Rhode Island	7	3	.15	53	.78	6	.09	79	1.16	27	.40		
South Carolina	636	615	6.61	820	4.33	66	.35	2,219	11.73	69	.36	5	.03
South Dakota	11	5	.23	29	.42	2	.03	51	.74	25	.36		
Tennessee	309	482	2.71	727	2.49	55	.19	1,577	5.39	361	1.23	9	.03
Texas	667	412	1.76	989	1.59	146	.23	2,740	4.40	846	1.36	87	.14
Utah	15	6	.40	50	.96	11	.21	82	1.57	45	.86		
Vermont	8		.21	12	.31	1	.03	22	.57	9	.23		
Virginia	377	331	2.53	772	2.81	68	.25	1,691	6.16	262	.96		
Washington	47	34	.48	88	.53	7	.04	192	1.15	231	1.38		
West Virginia	257	98	1.87	179	.94	30	.16	956	5.02	256	1.35		
Wisconsin	7		.02	80	.27	4	.01	91	.31	75	.25		
Wyoming	7	1	.34	8	.34	1	.04	25	1.05	18	.76	1	.04
Puerto Rico ⁴													
Virgin Islands ⁴													
Total	5,739	8,610	1.27	16,025	1.41	1,354	.12	40,304	3.54	12,036	1.06	265	.04

See footnotes at end of table.

Reports from cities of 200,000 population or over

	Syphilis								Gonorrhoea		Other venereal diseases		
	Early			Late		Congenital		All Syphilis		Number	Rate per 10,000 population	Number	Rate per 10,000 population
	Primary and secondary	Early-latent	Rate per 10,000 population	Includes late-latent	Rate per 10,000 population	Number	Rate per 10,000 population	Number	Rate per 10,000 population				
Akron.....	8	16	.87	29	1.05	6	.22	50	2.15	20	.73	-----	-----
Atlanta.....	-----	77	2.56	206	6.86	-----	-----	283	9.43	36	1.20	-----	-----
Baltimore.....	78	10	1.05	190	2.27	2	.02	610	7.30	195	2.33	15	.18
Birmingham.....	94	55	5.06	131	4.45	12	.41	385	13.08	36	1.22	1	.03
Boston.....	29	-----	.36	94	1.18	7	.09	162	2.04	131	1.65	-----	-----
Buffalo.....	10	1	.18	58	.96	-----	-----	69	1.15	39	.65	-----	-----
Chicago.....	83	228	.85	849	2.32	38	.10	1,198	3.27	851	2.32	40	.11
Cincinnati.....	-----	-----	-----	-----	-----	-----	-----	216	4.57	125	2.65	-----	-----
Cleveland.....	24	29	.56	124	1.31	13	.14	190	2.01	74	.78	-----	-----
Columbus.....	16	39	1.75	41	1.31	6	.19	102	3.25	27	.86	1	.03
Dallas.....	38	34	2.37	105	3.45	2	.07	179	5.89	134	4.41	19	.63
Dayton.....	18	9	1.22	32	1.44	1	.05	60	2.71	45	2.03	-----	-----
Denver.....	-----	-----	-----	-----	-----	-----	-----	142	4.71	68	2.26	-----	-----
Detroit.....	51	75	.69	418	2.30	13	.07	557	3.07	345	1.90	23	.13
Houston.....	35	31	1.84	167	4.66	24	.67	377	10.52	124	3.48	2	.06
Indianapolis.....	9	1	.26	18	.47	1	.03	114	2.96	35	.91	-----	-----
Jersey City.....	5	6	.34	25	.77	1	.03	37	1.14	9	.28	-----	-----
Kansas City ⁴	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----
Los Angeles.....	-----	128	.84	516	3.39	20	.13	664	4.36	442	2.91	4	.03
Louisville.....	21	4	.74	112	3.30	10	.30	147	4.34	305	9.00	3	.09
Memphis ⁴	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----
Milwaukee.....	2	-----	.03	57	.90	1	.02	60	.95	18	.29	18	.29
Minneapolis.....	7	26	.66	23	.46	-----	-----	65	1.30	45	.90	1	.02
Newark.....	54	-----	1.19	233	5.13	16	.35	393	8.65	90	1.98	-----	-----
New Orleans ⁴	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----
New York.....	281	326	.81	1,869	2.49	99	.13	2,809	3.75	1,202	1.60	11	.01
Oakland.....	6	9	.48	46	1.47	-----	-----	61	1.95	56	1.79	-----	-----
Omaha.....	13	4	.76	20	.89	1	.04	38	1.70	27	1.21	-----	-----
Philadelphia.....	229	631	4.29	-----	-----	32	.16	892	4.45	73	.36	-----	-----
Pittsburgh.....	-----	-----	-----	-----	-----	-----	-----	341	4.84	12	.17	-----	-----
Portland ⁴	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----
Providence.....	4	3	.27	32	1.23	1	.04	50	1.93	17	.65	-----	-----
Rochester.....	-----	-----	-----	16	.47	1	.03	17	.50	38	1.11	-----	-----
St. Louis.....	42	234	3.27	416	4.93	44	.52	736	8.73	195	2.31	11	.13
St. Paul.....	-----	-----	-----	-----	-----	-----	-----	30	1.04	20	.70	-----	-----
San Antonio ⁴	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----	-----
San Francisco.....	-----	-----	.75	242	3.51	8	.12	302	4.38	198	2.87	14	.20
Seattle.....	5	18	.59	68	1.76	4	.10	99	2.56	105	2.71	1	.03
Syracuse ⁴	-----	-----	-----	-----	-----	-----	-----	72	2.31	11	.35	2	.06
Toledo.....	4	6	.32	57	1.83	6	.19	-----	-----	-----	-----	-----	-----
Washington, D. C.....	-----	-----	-----	-----	-----	-----	-----	631	9.92	272	4.28	6	.09
Total.....	1,218	2,000	1.16	6,194	2.23	369	.13	12,147	4.03	5,420	1.80	172	.08

¹ Figures preliminary and subject to correction.

² Includes "not stated" diagnosis.

³ Duration of infection under 4 years.

⁴ No report for current month.

⁵ Includes early latent, late, and late latent.

WEEKLY REPORTS FROM CITIES

City reports for week ended May 18, 1940

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Small-pox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Data for 90 cities: 5-year average.....	134	84	37	5,998	558	1,973	20	393	26	1,234	-----
Current week 1.....	68	57	16	3,554	409	2,067	1	344	26	1,051	-----
Maine:											
Portland.....	0		0	100	0	0	0	0	0	1	17
New Hampshire:											
Concord.....	0		0	0	2	0	0	0	0	0	8
Manchester.....	0		0	0	1	0	0	0	0	0	11
Nashua.....	0		0	1	0	2	0	0	0	0	4
Vermont:											
Barre.....	0		0	0	0	0	0	0	0	6	10
Burlington.....	0		0	0	0	0	0	0	0	0	6
Rutland.....	0		0	0	0	0	0	0	0	0	6
Massachusetts:											
Boston.....	1		2	159	15	59	0	8	0	76	239
Fall River.....	3		0	55	1	1	0	0	0	4	30
Springfield.....	0		0	2	1	11	0	3	0	12	45
Worcester.....	0		0	107	1	0	0	0	0	6	46
Rhode Island:											
Pawtucket.....	0		0	1	0	0	0	0	0	0	15
Providence.....	1		1	118	4	6	0	1	1	9	61
Connecticut:											
Bridgeport.....	0		0	2	2	4	0	1	0	0	26
Hartford.....	0		0	1	4	18	0	2	0	0	42
New Haven.....	2		0	1	3	6	0	0	0	1	53
New York:											
Buffalo.....	2		0	5	9	17	0	4	0	2	148
New York.....	15	14	0	294	91	727	0	69	8	105	1,448
Rochester.....	0		0	7	4	17	0	0	1	7	76
Syracuse.....	0		0	0	1	9	0	0	0	0	43
New Jersey:											
Camden.....	2		0	1	3	13	0	0	0	0	32
Newark.....	0	2	0	460	8	35	0	0	0	15	105
Trenton.....	0		0	1	2	2	0	3	0	6	45
Pennsylvania:											
Philadelphia.....	2		0	145	19	123	0	27	0	32	479
Pittsburgh.....	1	2	1	3	15	24	0	4	2	15	168
Reading.....	0		0	1	0	1	0	4	0	8	29
Scranton.....	0		0	0		2	0		0	0	
Ohio:											
Cincinnati.....	1	1	0	4	5	17	0	4	0	17	96
Cleveland.....	0	11	2	6	12	44	0	16	0	34	203
Columbus.....	1	1	1	1	3	10	0	3	0	19	80
Toledo.....	0		0	4	3	43	0	1	1	11	61
Indiana:											
Anderson.....	0		0	0	1	0	0	0	0	3	6
Fort Wayne.....	0		0	3	1	0	0	0	0	0	28
Indianapolis.....	2		0	5	9	15	0	6	1	13	94
Muncie.....	0		0	0	2	0	1	0	0	0	21
South Bend.....	0		0	1	2	1	0	0	0	2	23
Terre Haute.....	0		0	0	0	2	1	1	0	1	19
Illinois:											
Alton.....	0		0	0	0	1	0	0	0	9	11
Chicago.....	2	2	2	99	28	483	0	36	0	37	711
Elgin.....	0		0	0	0	0	0	0	0	1	7
Moline.....	0		0	3	0	0	0	0	0	0	14
Springfield.....	0		0	0	2	4	0	0	0	0	26
Michigan:											
Detroit.....	5	1	0	216	11	108	0	12	2	92	297
Flint.....	0		0	8	7	20	0	0	0	10	20
Grand Rapids.....	0		0	6	1	29	0	0	0	18	40
Wisconsin:											
Kenosha.....	0		0	86	0	3	0	0	0	0	9
Madison.....	0		0	32	0	5	0	0	0	9	24
Milwaukee.....	0	1	1	120	4	34	0	2	0	1	106
Racine.....	0		0	1	0	7	0	1	0	2	9
Superior.....	0		0	105	2	4	0	0	0	0	8

¹ Figures for Barre estimated; report not received.

City reports for week ended May 18, 1940—Continued

State and city	Diph- theria cases	Influenza		Meas- les cases	Pneu- monia deaths	Scar- let fever cases	Small- pox cases	Tuber- culosis deaths	Ty- phoid fever cases	Whoop- ing cough cases	Deaths, all causes
		Cases	Deaths								
Minnesota:											
Duluth.....	0		0	11	2	1	0	1	0	0	24
Minneapolis.....	1		1	1	3	23	0	1	0	19	86
St. Paul.....	0		0	4	2	2	0	3	0	12	68
Iowa:											
Cedar Rapids.....	0			63		0	0		0	1	
Davenport.....	0			5		5	1		1	0	
Des Moines.....	0			11		9	1		0	0	31
Sioux City.....	1			2		3	0		0	0	
Waterloo.....	0			5		4			0	1	
Missouri:											
Kansas City.....	0		0	5	3	7	0	5	0	0	89
St. Joseph.....	0		0	0	0	0	0	1	0	0	23
St. Louis.....	1	2	0	8	3	20	0	5	1	12	215
North Dakota:											
Fargo.....	0		0	0	0	0	0	0	0	0	10
Grand Forks.....	0			0		0	0		0	0	
Minot.....	1		0	0	0	0	1	0	0	0	6
South Dakota:											
Aberdeen.....	0			0		0	0		0	0	
Sioux Falls.....	1		0	0	0	2	0	0	0	0	8
Nebraska:											
Lincoln.....	0			1		3	0		0	2	
Omaha.....	4		0	5	3	0	0	3	0	3	61
Kansas:											
Lawrence.....	0		0	0	0	0	0	0	0	0	3
Topeka.....	0		0	21	4	2	0	1	0	1	25
Wichita.....	0	1	0	3	4	0	0	0	0	5	23
Delaware:											
Wilmington.....	0		0	0	1	1	0	0	0	0	30
Maryland:											
Baltimore.....	0	2	1	3	13	14	0	10	0	119	214
Cumberland.....	0		0	0	0	0	0	0	0	0	18
Frederick.....	0		0	0	0	0	0	1	0	0	6
Dist. of Col.:											
Washington.....	2		0	3	10	33	0	8	0	5	173
Virginia:											
Lynchburg.....	0		0	2	0	0	0	0	0	5	9
Norfolk.....	0	8	0	16	2	2	0	0	1	0	16
Richmond.....	0		0	4	4	6	0	2	0	3	47
Roanoke.....	0		0	18	0	1	0	0	0	0	14
West Virginia:											
Charleston.....	0	1	0	0	1	0	0	0	1	0	28
Huntington.....	2			0		4	0		0	0	
Wheeling.....	0			1		0	0		0	4	
North Carolina:											
Gastonia.....	0			0		0	0		0	0	
Raleigh.....	0		0	0	0	0	0	1	0	5	6
Wilmington.....	0		0	1	0	1	0	1	0	0	12
Winston-Salem.....	0		0	0	0	1	0	1	0	2	19
South Carolina:											
Charleston.....	0	3	0	0	3	0	0	2	0	0	24
Florence.....	0		0	0	0	0	0	0	0	0	4
Greenville.....	0		0	0	6	0	0	0	0	0	21
Georgia:											
Atlanta.....	0		0	6	6	3	0	9	0	0	92
Brunswick.....	0		0	1	0	1	0	1	0	0	3
Savannah.....	0	1	0	2	1	1	0	3	2	0	30
Florida:											
Miami.....	0	1	1	1	1	1	0	2	0	0	35
Tampa.....	0	1	0	48	1	1	0	4	0	1	15
Kentucky:											
Ashland.....	0		0	16	1	0	0	0	0	12	8
Covington.....	0		0	8	0	1	0	1	0	0	14
Lexington.....	0		0	24	1	1	0	1	0	3	16
Louisville.....	0		0	6	3	25	0	4	0	73	86
Tennessee:											
Knoxville.....	1	1	0	11	2	8	0	1	1	1	31
Memphis.....	0	1	0	15	3	12	0	3	2	19	74
Nashville.....	0		0	10	2	2	0	3	0	15	40
Alabama:											
Birmingham.....	2	2	0	13	7	3	0	5	3	1	61
Mobile.....	0		0	0	0	0	0	2	0	0	21
Montgomery.....	0			2		0	0		0	4	

City reports for week ended May 18, 1940—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Small-pox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Arkansas:											
Fort Smith.....	0			0		0	0		0	0	
Little Rock.....	0	1	0	0	2	0	0	1	0	1	
Louisiana:											
Lake Charles.....	0		0	2	1	0	0	0	0	0	6
New Orleans.....	7	1	1	3	13	5	0	8	2	22	147
Shreveport.....	1		0	0	4	0	0	0	0	3	32
Oklahoma:											
Oklahoma City.....	0	3	0	0	3	3	0	1	0	0	45
Texas:											
Dallas.....	2		0	650	2	1	0	4	0	20	56
Fort Worth.....	0		0	12	0	0	0	2	0	36	44
Galveston.....	0		0	0	0	1	0	1	0	0	18
Houston.....	3		1	10	9	0	0	4	0	4	69
San Antonio.....	0		0	5	5	0	0	10	0	0	77
Montana:											
Billings.....	0		0	0	1	0	0	0	0	0	7
Great Falls.....	0		0	21	1	2	0	0	0	0	11
Helena.....	0		0	0	0	0	0	0	0	0	
Missoula.....	0		0	0	0	0	0	0	0	0	11
Idaho:											
Boise.....	0		0	2	0	2	0	0	0	0	5
Colorado:											
Colorado Springs.....	0		0	3	0	0	0	0	0	0	8
Denver.....	4		2	34	2	8	0	4	0	1	70
Pueblo.....	0		0	6	1	5	0	2	0	0	12
New Mexico:											
Albuquerque.....	0	1	0	0	1	0	0	3	0	2	13
Utah:											
Salt Lake City.....	0		0	260	2	5	0	0	0	116	30
Washington:											
Seattle.....	0		0	207	3	5	0	1	0	17	92
Spokane.....	0		0	8	0	4	0	0	0	1	29
Tacoma.....	0		0	1	1	4	0	0	0	0	32
Oregon:											
Portland.....	0		0	83	0	6	0	0	0	6	51
Salem.....	0			2		0	0		0	0	
California:											
Los Angeles.....	1	6	0	22	7	23	0	22	0	56	353
Sacramento.....	0		0	11	1	5	0	1	0	17	20
San Francisco.....	0		0	5	7	8	0	0	3	21	171

State and city	Meningococcus meningitis		Polio-myelitis cases	State and city	Meningococcus meningitis		Polio-myelitis cases
	Cases	Deaths			Cases	Deaths	
New York:				Louisiana:			
Buffalo.....	3	3	0	Shreveport.....	0	1	0
New York.....	1	0	1	Utah:			
Pennsylvania:				Salt Lake City.....	0	0	1
Pittsburgh.....	1	0	0	Washington:			
Ohio:				Tacoma.....	0	0	2
Cleveland.....	0	0	1	California:			
Maryland:				Los Angeles.....	0	0	2
Baltimore.....	1	0	0				
West Virginia:							
Huntington.....	0	1	0				

Encephalitis, epidemic or lethargic.—Cases: New York, 1; Alton, 1; Helena, 1.
Pellegra.—Cases: Boston, 1; Atlanta, 1; Los Angeles, 1.
Typhus fever—Cases: New York, 1.

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended May 4, 1940.—
 During the week ended May 4, 1940, cases of certain communicable diseases were reported by the Department of Pensions and National Health of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Cerebrospinal meningitis.....		1	1	5	1			1		9
Chickenpox.....		1	2	101	233	33	5	15	59	449
Diphtheria.....				23		6	1			30
Influenza.....		10			94				6	110
Lethargic encephalitis.....				2			1			3
Measles.....		7		186	343	514	375	3	73	1,501
Mumps.....				26	278	10	21		14	349
Pneumonia.....	5	3			30		4		8	50
Scarlet fever.....			3	102	155	16	4	23	5	308
Tuberculosis.....	6	2	26	53	59	4	4	1		155
Typhoid and paratyphoid fever.....		1		30	12					43
Whooping cough.....		29	2	147	128	39	68		21	434

CUBA

Provinces—Notifiable diseases—4 weeks ended April 27, 1940.—
 During the 4 weeks ended April 27, 1940, cases of certain notifiable diseases were reported in the Provinces of Cuba as follows:

Disease	Pinar del Rio	Habana	Matanzas	Santa Clara	Camaguey	Oriente	Total
Cancer.....	1	2		6	2	5	16
Chickenpox.....		2		2	9	2	15
Diphtheria.....		10	1	3		3	17
Hookworm disease.....				1			1
Leprosy.....			1	1		1	3
Lethargic encephalitis.....		1					1
Malaria.....	12			3		23	38
Measles.....	2	8	6	2	2	9	29
Meningitis, tubercular.....		1					1
Poliomyelitis.....				1		1	2
Tuberculosis.....	27	39	16	26	12	21	141
Typhoid fever.....	17	129	5	39	15	40	245

SWITZERLAND

Notifiable diseases—March 1940.—During the month of March 1940, cases of certain notifiable diseases were reported in Switzerland as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis.....	138	Mumps.....	72
Chickenpox.....	109	Paratyphoid fever.....	3
Diphtheria.....	45	Poliomyelitis.....	5
German measles.....	125	Scarlet fever.....	398
Influenza.....	2,498	Tuberculosis.....	224
Lethargic encephalitis.....	4	Undulant fever.....	11
Measles.....	1,600	Whooping cough.....	197

REPORTS OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER RECEIVED DURING THE CURRENT WEEK

NOTE.—A cumulative table giving current information regarding the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS of May 31, 1940, pages 1000-1002. A similar table will appear in future issues of the PUBLIC HEALTH REPORTS for the last Friday of each month.

Smallpox

China.—During the week ended May 4, 1940, 1 case of smallpox was reported in Shanghai, 1 case in Hong Kong, and 23 cases in Macao, China.

Thailand—Bangkok.—During the week ended May 4, 1940, 4 cases of smallpox, with 1 death, were reported in Bangkok, Thailand.

Typhus Fever

Japan—Tokyo.—On April 26, 1940, a case of typhus fever was reported in Tokyo, Japan.